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Physiological and Psychological Markers of Stress Predicting Psychopathology in First Responders

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Physiological and Psychological Markers of Stress Predicting Psychopathology in First Responders

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Dedication

This dissertation is dedicated to my grandparents, Dr. Sheldon Joseph & Betty Joseph. I also dedicate this work to Jennifer Malin; she was an essential member of my doctoral dissertation study and a friend whom is missed dearly. Finally, I dedicate this dissertation to the local emergency medical service personnel participants whom allowed us a glimpse of their lives at work and off-the-job.

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Physiological and Psychological Markers of Stress Predicting Development of Psychopathology in First Responders

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While there exists a small but growing literature on the effects of stress and mental health prevalence for emergency first responders, a paucity remains for first responder research focusing on the pathogenic effects of the stress response that incorporates both traditional psychosocial measures of stress and biological markers of stress measured by salivary endocrine levels for the stress-linked hormones cortisol and testosterone. Stress research in the social sciences has overwhelmingly evidenced the allostatic effects of cortisol and testosterone in the human and animal stress response. The cross-talk between the two hormone pathways when an individual perceives stress affects mental health and lends growing support for investigation of the dual-hormone hypothesis of cortisol and testosterone in models of psychopathology. Psychological and physiological stress variables were measured at baseline for a cohort of local emergency first responders (N=190). Traditional stress-buffering (stress-protective) psychosocial constructs of social support and resiliency were also measured. Symptoms of mood, anxiety, and trauma-related disorders, alcohol use, and sleep quality were assessed at baseline, but also, at 3-month and 6-month follow-up for each participant. Ordinary least square (OLS) linear regression was used to predict if hormone biomarkers and self-reported baseline perceived stress were associated with change in clinical symptoms at 3-month (N=158) and 6-month

follow-up (N=111). No single nor dual hormone effects of cortisol or testosterone were supported within the data as diatheses for stress-linked psychopathology. High numbers of models corrected for by adjusting *p* values and small sample size are likely implicated in null findings. In addition, study limitations are discussed regarding calculation of prediction models using OLS regression rather than multi-level modeling regression. The addition of further refined endocrine and psychosocial stress variables is discussed for future studies. This study contributes a comprehensive literature review of the first responder stress literature and a novel investigation of the dual-hormone hypothesis within a population of first responders.

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Chapter 1: Literature Review

Emergency first responders—including paramedics or ambulance personnel, firefighters, emergency room medical staff, disaster and aid workers, and law enforcement—are subject to high levels of job-related stress (Kleim & Westphal, 2011). First responders witness events involving human suffering at a higher frequency than the general population (van der Ploeg & Kleber, 2003; Gayton & Lovell, 2012; Kleim & Westphal, 2011; Regehr, Goldberg, & Hughes, 2002). They typically arrive to an emergency with the expectation of attending to injured victims, retrieving deceased victims, and working with survivors and their families (Kleim & Westphal, 2011). Haugen, Evces, and Weiss (2012) argue that only two professions directly and repeatedly expose their employees to chronic stress and potential traumas: the military and first responder services. Unfortunately, the amount of research on stress among first responders pales in comparison to the number of studies involving military personnel or veterans (Haugen et al., 2012; Kleim & Westphal, 2011).

In order to develop more effective preventative care and treatments for this special at-risk population, we first need to understand the biological and psychosocial mechanisms underlying stress and trauma, and the consequent links to stress-linked psychopathologies (e.g., PTSD, depression, anxiety). This review starts with an in-depth investigation in the endocrine system's involvement in the human stress response, focusing in particular on the research-indicated hormones cortisol and testosterone, and discusses the putative mechanisms of these hormones influencing stress-linked psychopathology. Following the hormone literature, the effects of perceiving oneself to be 'stressed' supplement the stress background sections. These commonly-deemed 'psychosocial' factors have direct influence on the aforementioned hormonal systems often considered primarily 'biological'

components or physiological markers of stress. In the same vein, research into the perceptions of interpersonal support and intrapersonal resiliency are presented as they are also shown to influence the human physiology of the stress reaction. Considering the significant stress-related factors presented in this review, this dissertation study is presented within a diathesis-stress framework, a theoretical framework design used to understand the moderating interactions between these endocrine and psychosocial factors affecting mental illness. This review will reinforce the idea that hormones interact with stress to influence the stress-linked psychopathology. Stress-buffering factors, such as resiliency and social support, are also associated with the hormone-stress interaction. This interactive effect of stress and coping factors allows downstream predictions of an individual's mental health and risk of psychopathology. This chapter review then returns to first responder-specific research literature to describe the hormonal diathesis-stress models and moderators that have been studied in the first responder population to date.

THE STRESS RESPONSE: PHYSIOLOGICAL FACTORS

The history of stress research dates nearly as far back as the science of psychology itself. In the mid-to-late 1800s, neurologist George M. Beard popularized the term "neuroasthenia"—or nervous exhaustion—as he believed that the nervous system had the potential to lose nutrients and required "air, sunlight, water, food, rest, diversion, muscular exercise, and the administration of strychnine, phosphorus, and arsenic" to be replenished (Jackson, 2013, p. 26). While Beard misidentified the last three nutrients, which in reality can be neurotoxic, he positively identified protective health factors for physical and mental health; he was an early advocate for describing how individuals' minds were sensitive to physical and environmental conditions. Subsequent researchers studied the physiological

underpinnings of these "environmental conditions," which came to be recognized as "stress." In foundational physiological studies, Walter Cannon identified a physiological reaction to stress within the body in the 1920s and 1930s. Throughout his work, he highlighted how emotions and environmental conditions change the physiological balance within the body – particularly within what we now know as the autonomic nervous system (Cannon, 1932). He determined that the preferred physiological status of the body is to be non-reactive to these emotions and environmental stress; however, if reactive to its environment or emotions, the body would provide itself feedback to direct its return to its preferred non-reactive status – a state Cannon called "homeostasis" (Cannon, 1929; Quick & Spielberger, 1994).

Following up on Cannon's ground-breaking work, Selye expounded upon the stress-homeostasis theory by noting that animals and humans have similar physiological reactions to these activating (usually unpleasant) environmental conditions (1936; 1950). Adapting a term from physics, Selye called these antecedents of the body's response "stress" because they act upon the body as a force. Importantly, Selye developed the theory of General Adaptation Syndrome (GAS), which explained how stress activates a chain-reaction within the neuroendocrine system across the body, extending from the body's glands placed in the brain all the way to kidneys (1956; Cohen, Gianaros, & Manuck, 2016). He noted that this same chain reaction is activated in response to a broad array of stressors (Selye, 1956; Sapolsky, 2004), and described the stress response as non-specific (i.e., "many different stressors elicit a similar stress response") (Nelson, 2005, p. 672). We now know GAS as the stress response system.

Cortisol

The stress response system responds primarily through a main neuroendocrine axis called the hypothalamic-pituitary axis (HPA). Stress acts on the individual, and as the

individual perceives and appraises this stress, the brain secretes a hormone called corticotropin-releasing hormone (CRH) from the hypothalamus. CRH circulates to the anterior pituitary gland in the midbrain and activates the release of adrenocorticotropic hormone (ACTH). ACTH then circulates to the adrenal glands of the kidneys where it's absorption results in the renal secretion of cortisol—a steroid glucocorticoid hormone that serves as the end product of this hormone-releasing axis (Figure 1; Miller & O'Callaghan, 2002). However, this multi-step chain reaction does not continue indefinitely once initiated. As cortisol is released into circulation, the glucocorticoid is reabsorbed via receptors on the anterior pituitary gland as well as the hypothalamus; this reabsorption acts as the mechanism for negative feedback within the system (Miller & O'Callaghan, 2002; Nelson, 2005). The release of hormones precipitates the body's natural shut down of the stress reaction by reabsorption.

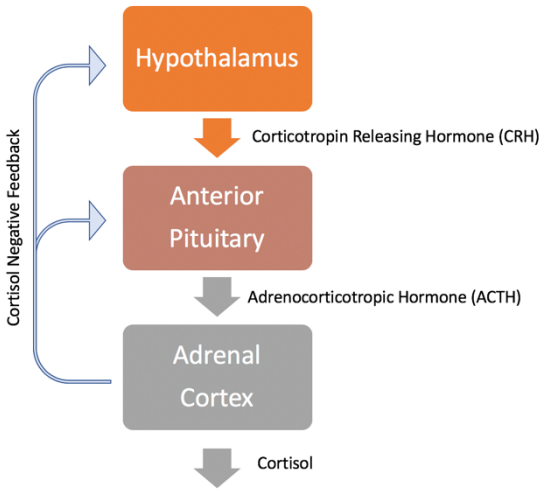

Figure 1: Hypothalamic-pituitary axis stress response.

Contemporary research has continued the field's tradition into the physiological effects of stress (or a stressor) upon the body's response but is often supplemented by accounting for psychosocial individual differences in the face of stress for a greater behavioral understanding of the response. Acute stress is defined as "the recognition by the body of a stressor and therefore, the state of threatened homeostasis; stressors are threats against homeostasis; and the adaptive responses are the body's attempt to counteract the stressor and reestablish homeostasis" (Nelson, 2005, p. 679; Chrousos & Gold, 1992). Although the term 'stress' often carries a negative connotation, Sapolsky defines a stressor as anything that disrupts the body's homeostasis, whether the individual appraises this disruption as positive or negative (2004; Kagan, 2016).

This purely physiological concept of stress and homeostasis is problematic, however, as it does not align with the majority of psychological stress research done in humans. In practice, human stress research typically addresses not only physiological changes within an individual but also psychological perception of a stressor to account for individual variation in a stressed environment (Nelson, 2005). Although the HPA axis can be activated by either pleasurable or negative events, the majority of stress research conceptualizes stressors as negative. For example, Kim and Diamond (2002) theorized that when an individual is aroused by an aversive stimulus, the magnitude of the stress is then determined by the individual's perception of control over the aversive stimulus. If perceived as aversive, the arousal is deemed "stress." The present study is based on this multi-faceted conceptualization of stress – including physiological change as well as

one's own psychological perception of the stressor, which is frequently used across the field of stress research.

The stress response promotes adaptation. The continued heritability of this system has likely persisted because the acute stress response helps individuals cope with emergency situations (Nelson, 2005). For example, in the wild, a zebra's perception of threat and fear physiologically prompts that zebra to run from a predatory lion. The release of cortisol activates the body through mechanisms including increased cardiovascular tone, respiration rate, and blood flow to the muscles from the trunk (Nelson, 2005). The notion that acute stress can generate adaptive reactions led researchers to conceptualize the fight-or-flight response (Cannon, 1929) and, later, the tend-and-befriend response (Taylor et al., 2000) as economical strategies for health and survival, given an animal's perceived physiological and psychological resources. Taylor et al. (2000; Taylor, 2006) posit that hormonally-driven reproductive demands lead to sexually dimorphic behavioral adaptations to stress challenges to stress. While both female and male animals respond physiologically to the stressor via the HPA axis, the male may more commonly provide flight-or-fight behavior responses while the female may be more defensive in her strategies to combat the stressor by nurturing activities and herd protection strategies to protect offspring (Taylor, 2006). This differential is also observed in human stress research (Turton & Campbell, 2005; Tamres, Janicki, & Helgeson, 2002). However, studies of stress research, particularly human stress studies, have shown that gender lines are not strictly drawn as males and females are both capable of affiliative responses (or prosocial behavioral) in response to acute stress (Von Dawans,

Fischbacher, Kirschbaum, Fehr, & Heinricks, 2012). To conclude, regardless of sex, animals including humans are known to physiologically and behaviorally react to the stressor to foster evolutionary, biological, psychological, and social adaptation.

Although stress can be a positive adaptive force for survival, it can also lead to pathophysiological consequences within the individual. Whereas HPA activation, culminating in elevated circulating cortisol levels, is a cardinal physiological response to acute stress (Michaud, Matheson, Kelly, & Anisman, 2008), if stress persists, the result can be chronically-elevated cortisol, which can cause excessive wear on the efficacy of the HPA downstream reaction and damage areas of the brain associated with memory and emotion regulation (Miller, Chen, & Zhou, 2007). Selye noted early on that repeated exposure to stressors could result in atrophy of the adrenal glands, thalamic atrophy, and gastrointestinal distress over time (1936). Therefore, the general consensus is that differentiation across acute and chronic stress depends on the temporal nature of the stressor(s) and the adaptive or maladaptive nature of the individual's physical and psychological response. Over time, chronic stressors result in chronic negative appraisals of threat and frequent activation of the HPA response (Nelson, 2005; Sapolsky, 1992). Research has shown that the long-term repetition of this cycle results in disease-related physiological changes and increased risk of disease onset or progression (Figure 2; Cohen et al., 2016). Whereas the stress response is adaptive in the short term, prolonged stress is considered maladaptive because it activates prolonged stress responses within the body. McEwen proposed that this chronic (frequent and/or prolonged) activation of the HPA axis-directed stress response created a "wear-and-tear" on the body, which he

termed “allostatic load.” Physiological manifestations of this allostatic load include: suppressed immunity, atherosclerosis, obesity, bone demineralization, and nerve degeneration (2004; Cohen, 2004).

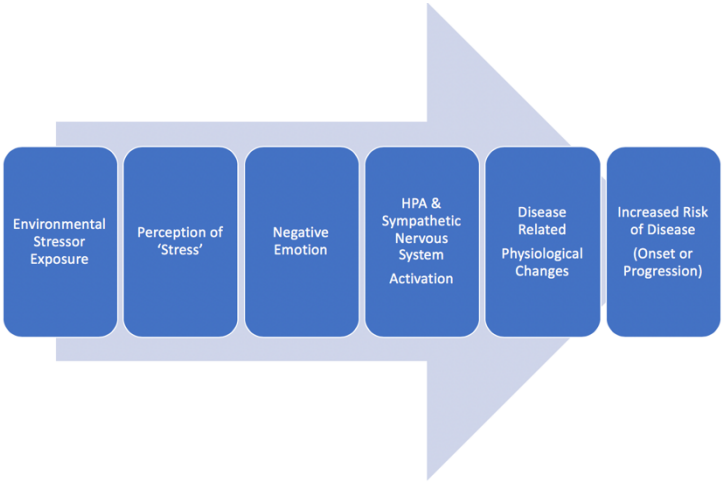

Figure 2: Theoretical model of maladaptive chronic stress related to disease.

Cortisol and Psychopathology

Excessive cortisol can lead to Cushing’s disease with increased facial and abdominal weight gain, hypertension, irritability, insomnia, memory and concentration difficulties, edema, diabetes, depression, irritability and other personality changes (Kirk, Hash, Katner, & Jones, 2000). On the other hand, a deficiency of cortisol can lead to Addison’s disease, an autoimmune disease whose symptoms include confusion, lethargy, circulatory depression, and risk of death (Michels & Michels, 2014). Even when dysregulation of the neuroendocrine system is at a severity lesser than required for clinical diagnoses of these diseases, mild dysregulation can still result in adverse health consequences. After treatment of these problematic physiological symptoms (i.e., too high

or too low cortisol levels), physicians noticed simultaneous improvement of mental health or psychological functioning (Sonino, Fava, Raffi, Boscaro, & Fallo, 1998). Of note, researchers who treated men with Cushing's disease with dexamethasone—a cortisol antagonist due to its nature as a synthetic glucocorticoid – observed improvements in mood (Heuser, Yassouridis, & Holsboer, 1994). These findings and others led to the suggestion that hyperactivity of the HPA and excessive cortisol production might be significant risk factors in the pathogenesis of depression (Holsboer, 2000; Nemeroff & Vale, 2005; Plotsky, Owens & Nemeroff, 1998; Schlesser, Winokur & Sherman, 1980). In support of this, the dexamethasone suppression test – typically used to identify individuals with HPA axis tumors – was used to diagnose cortisol dysregulation as a risk factor for depression. Not surprisingly, in light of evidence linking cortisol to psychopathology, HPA axis dysregulation has since been linked to mood (Burke, Davis, Otte, & Mohr, 2005) and anxiety disorders (Jezova, Makatsori, Duncko, Moncek, & Jakubek, 2004). Research on connections between PTSD and HPA dysregulation has produced mixed findings. Several studies show lower or flattened cortisol levels (or hyporegulation of the HPA) in individuals with PTSD (Meewisse, Reitsma, de Vries, Gersons, & Olff, 2007; Lauc, Zvonar, Vukšić-Mihaljević, & Flögel, 2004; Yehuda, 2001); however, other studies have reported high cortisol levels in those with moderate and severe PTSD symptoms (Violanti et al., 2007). Given these evidence-based findings between cortisol and psychopathology on which this dissertation is based, the present study will focus on the effects of cortisol levels within the sample population on mental health outcomes such as depression, anxiety, and PTSD as well as psychopathology-related health behaviors (sleep and substance use).

Testosterone

Testosterone is a steroid hormone from the androgen class of hormones, best known for its influences on mating and sexual behavior. It is produced in the Leydig cells of the testes of men and in the adrenal glands of both men and women (in women's ovaries, specific enzymes convert testosterone to estrogen via a process called aromatization) (Nelson, 2005). Testosterone is the main end-product of the hypothalamic-pituitary gonadal axis (HPG), just as cortisol is the end product of the HPA. The androgen class of hormones has a varied array of physiological and behavioral functions. The presence of testosterone is responsible for sex differentiation of the primary sex organs and secondary sex characteristics in males (Nelson, 2005). Before testosterone had been identified, early experimental research with animal castration (using roosters) found that testosterone's effects were not restored by re-transplanting the testes; this finding led researchers to conclude that testosterone exerts its behavioral and sexual characteristics via secretion into the bloodstream (Berthold, 1849; Freeman, Bloom, & McGuire, 2001; Nelson, 2005). The development of synthetic testosterone in the twentieth century to treat hypogonadal men found significant increases in sexual drive (Hoberman & Yesalis, 1995).

Testosterone also has been found to affect the stress response in a generally protective manner, reducing allostatic load on the HPA (McEwen, 2000). The putative mechanism of this adaptive response is that testosterone functions to reduce the danger of a perceived threat by increasing a stimulus's reward value and decreasing its punitive value (Korte, Koolhaas, Wingfield, & McEwen, 2005). Research in psychoneuroendocrinology has shown that stress is associated with reduced testosterone production via several mechanisms. These include suppression of gonadotropin-releasing hormones and CRH receptors indirectly in the HPG pathway as well as reduction of testosterone in the Leydig cells when enzymes in the testes, which usually neutralize glucocorticoids, are

overwhelmed by prolonged glucocorticoid production due to prolonged stress (Roy, Kirschbaum, & Steptoe, 2003). Controlling for age, studies have found that daily stress over time results in decreased testosterone levels (Roy et al., 2003).

Testosterone and Psychopathology

If stress suppresses testosterone levels, it follows that individuals with higher levels of baseline testosterone might be better protected from the negative physiological and psychopathological effects of stress's allostatic load. Research has shown that age-controlled males with higher psychological stress had significantly lower testosterone levels than their lower-stress counterparts (Francis, 1981). Perception of a potential threat to one's health from grave illness was rated lower among individuals with higher testosterone levels (Ristvedt, Josephs, & Liening, 2012). As perception of threat has a strong positive association with emotion regulation and mood, higher testosterone is related to more positive mood (Booth, Johnson, & Granger, 1999).

Additionally, studies of anxiety and depression disorders in males have found that higher testosterone yields protective benefits against anxiety and depression symptoms (McHenry, Carrier, Hull, & Kabbaj, 2014). Exogenous administration of testosterone in hypogonadal men leads to significant increases in mood (Wang et al., 2000; Jockenhövel et al., 2009). Clinical evidence suggests that exogenous testosterone has anxiolytic and antidepressant benefits for women as well (Goldstat, Briganti, Tran, Wolfe, & Davis, 2003; Hermans, Putman, Baas, Koppeschaar, & van Honk, 2006). In a congruent laboratory study, the administration of exogenous testosterone was shown to reduce gaze aversion in women with social anxiety disorder leading to improved eye contact, which is commonly impaired within those with the disorder (McHenry et al., 2014). The neurobiological mechanisms of these benefits are not clearly understood, but they are likely connected to

testosterone's modulatory effects on anxiety and depression-related neurotransmitters such as activation of dopamine and serotonin, and inhibitory activation of GABA (McHenry et al., 2014; Bitran, Kellogg, & Hilvers, 1993).

Synergetic Effects of Cortisol and Testosterone – The Dual Hormone Hypothesis

Given the involvement of both cortisol and testosterone in the stress response, relatively few studies have looked into the synergetic effects of both hormones in the scientific literature, but those studies to date have found significant cross-talk between these two hormone axes. Viau and Meaney found that testosterone can attenuate high cortisol levels (1996). Combined with established evidence of testosterone's neuroprotective effects on allostatic load, more research on the dual-effect is warranted to examine testosterone in the context of cortisol regulation and is therefore included in this dissertation study.

A small but growing body of research has investigated multiple hormone interactions as physiological factors in in the etiology of stress-linked behaviors. Supporting evidence for the joint effects of cortisol and testosterone (termed "the dual hormone hypothesis" by Mehta and Josephs, 2010) comes from studies on competition and dominance (Mehta & Josephs, 2010; Liening & Josephs, 2010), social performance (Bedgood, Boggiano, & Turan, 2014), and risk-taking (Mehta, Welker, Zilioli, & Carré, 2015). For a review of social behaviors tested using the dual-hormone hypothesis see Mehta & Prasad for a comprehensive review (2015). The majority of the evidence in support of the dual hormone hypothesis studies has shown that high testosterone is associated with greater social approach behaviors or status achievement, but only when cortisol levels are low (Mehta & Josephs, 2010; Mehta & Prasad, 2015). However, Mehta & Prasad acknowledge two studies have shown that *low* testosterone is associated with

social approach behaviors when cortisol is low (2015). Although the putative mechanisms of this cortisol-testosterone cross-talk have not been fully fleshed-out, one explanation for the testosterone by cortisol interaction is suggested by testosterone's suppression of HPA axis activity (Mehta & Josephs, 2010; Sapolsky, 2004; Viau & Meaney, 1996; Viau, 2002).

More recent studies have extended the dual hormone hypothesis to investigate the joint role of testosterone and cortisol in psychopathology, including externalizing psychopathology (Tackett, Herzhoff, Harden, Page-Gould, & Josephs, 2014), PTSD (Josephs, Cobb, Lancaster, Lee, & Telch, 2017) and depression (Cobb, Josephs, Lancaster, Lee, & Telch, 2018). The evidence for the dual effects of testosterone and cortisol on psychopathology follows in the same vein as the dual hormone effects on social behaviors. The single-hormone literature has reported that higher testosterone levels are generally protective against negative mood and anxiety (Maeng & Milad, 2015; McHenry et al., 2014; Zarrouf, Artz, Griffith, Sirbu, & Kommor, 2009; Zitzmann, 2006). However, the PTSD literature is more mixed. Some PTSD studies report that subjects have lower levels of testosterone than healthy controls (Mulchahey et al., 2001; Gomez-Merino et al., 2005; Fenchel et al, 2015), whereas other studies of PTSD patients have shown testosterone to be elevated in PTSD patients compared to healthy controls (Reijnen, Geuze, & Vermetten, 2015; Karlovic et al., 2012; Yehuda, 1998; Mason, Giller, Kosten, & Wahby, 1990). Testosterone's role as a biomarker for PTSD is not clear as these incompatible results may be due to different populations and different measurement periods following trauma. Moreover, these PTSD studies did not consider cortisol levels as a moderator of the association between testosterone and PTSD symptoms.

Given this lack of clarity in the connections between testosterone and psychopathology, and the lack of clarity in the cortisol-psychopathology literature, with some studies pointing to high cortisol as a risk factor for depression (Holsboer, 2000; Burke

et al., 2005; Gold, Drevets, & Charney, 2002; Goodyer, Herbert, Tamplin, & Altham, 2000), with other studies pointing to blunted cortisol levels as pathogenic (Meewisse et al., 2007; Lauc et al., 2004), further investigation into the joint role played by testosterone and cortisol in psychopathology risk seems overdue.

Yet another question that merits consideration in hormone studies of psychopathology is the role of basal hormone levels versus stress-evoked hormone changes, or *reactivity*. Josephs et al. (2017) measured cortisol and testosterone *reactivity* in a group of active military members to a perceived threat, and then assessed exposure to in-theatre warzone stressors to predict changes in PTSD symptoms during deployment. Soldiers' cortisol and testosterone levels were measured prior to and after exposure to a carbon dioxide (CO_2) challenge. This challenge can produce dizziness, light-headedness, vertigo, fear of suffocation, and panic, presumably due to the decreased oxygen content (van den Hout & Griez, 1984). Josephs et al. (2017) reported that soldiers with low cortisol reactivity and low testosterone reactivity interacted to predict war-zone-stress-evoked PTSD symptoms during deployment. Increased levels of PTSD symptoms in the face of war-zone stressors was highest among soldiers with low cortisol reactivity *and* low testosterone reactivity. Conversely, soldiers with low cortisol reactivity *but* high testosterone reactivity did not show war-zone-stress-evoked increases in PTSD. War-zone-stress-evoked levels of PTSD were not predicted by basal hormone levels. This result argues for the importance of hormonal reactivity. Unfortunately, this dissertation study did not include an acute stress exposure.

In a study of war-zone-stress-evoked depression symptoms using the same sample as used in Josephs et al. (2017), Cobb et al. (2018) reported that cortisol and testosterone (both basal and reactivity levels), individually, were associated with the presence of clinical depression symptoms in active military during deployment. However, no evidence of dual

hormone effects on war-zone-stress-evoked depression symptoms were reported. Cobb et al. (2018) reported that low basal cortisol levels predicted depression, whereas high testosterone levels protected against depression.

In other behavioral health-related disorders, testosterone and cortisol have been examined as factors in risk for psychopathy (Glenn, Raine, Schug, Gao, & Granger, 2011), adolescents at risk for externalizing disorders (Tackett et al., 2014), and overt aggression (Popma et al., 2007). Glenn et al. (2011) found a higher ratio of basal testosterone to cortisol reactivity was predictive of psychopathic traits. Tackett et al. (2014) found that high testosterone was associated with greater externalizing behaviors but only when cortisol was low. Popma et al. (2007) found that high testosterone was predictive of aggressive behaviors in individuals only when cortisol was low.

Results of dual and single hormone effects from Josephs et al. (2017) and Cobb et al. (2008), respectively, and other dual hormone studies of psychopathology suggest that we may expect to find dual hormone effects in first responders and other populations that are regularly exposed to potentially traumatic stressors. However, with the exception of the current study, research has yet to investigate the joint effects of cortisol and testosterone on mental health outcomes among first responders. Although evidence for a risky cortisol profile (high vs. low basal cortisol) is mixed in the literature for the outcomes measured in this dissertation study, hypercortisolism's allostatic load on physical health, and its correlation with decreased mood and increased anxiety support the hypothesis that high cortisol levels will be associated with increased levels of psychopathology in this understudied population.

THE STRESS RESPONSE: PSYCHOLOGICAL FACTORS

The individual's own perception of "stress" is also associated with the stress response by directly influencing the physiological changes the body undergoes. Perceived stress is associated with increased cortisol secretion (Schlotz, Hammerfald, Ehlert, & Gaab, 2011); as noted above, cortisol is the end product of the body's HPA endocrine axis. Similar to the discussion of an acute or chronic stressor's effect on the HPA endocrine response, the individual's own perception of the stressor to be acute (i.e., rapid-onset, short-term) or chronic (long-term, undetermined duration and frequency) creates its own effect on the stress system over and above the presence of the stressor. Chronic perceived stress is also associated with increased cortisol production, but most interestingly, the magnitude of the stress response is dependent on whether the stressor is still ongoing and how frequently it has occurred for the individual (Van Eck, Berkhof, Nicolson, & Sulon, 1996). Stressors perceived to be ongoing with no foreseen end and those stressors anticipated to be repeated in the future are greater risks for cortisol dysregulation. It has been shown that higher perceived stress levels over time have been linked to greater dysregulation of the stress response compared to their non-chronically stressed peers (van Eck et al., 1996; Miller & O'Callaghan, 1991).

It logically follows that if perceived stress is associated with physiological changes (i.e. dysregulation due to allostatic load) in the stress response, which in turn, is linked to risk of psychopathology, then perceived stress is also an associated risk factor for psychopathology. When an individual perceives that they do not have adequate adaptability to mitigate a stressor (i.e., a physiological and psychological return to homeostasis), the stressor elicits downstream emotional responses including worry, fear, and anxiety (Brosschot, Gerin, & Thayer, 2006; Miller & O'Callaghan, 2002). The intensity and frequency of these emotional responses in reaction to one's appraisal of limited adaptability

to the stressor at hand creates a greater risk of persistent negative mood states such as sadness, hopelessness, anxiety and worry, and depression (Brosschot et al., 2006). These negative cognitions and mood states are key cognitive-affective features of mood-based psychopathologies including depression and anxiety, and trauma-based PTSD (Beck & Bredemeier, 2016; Disner, Beevers, Haigh, & Beck, 2011 for depression; Barlow, 2000; Eysenck & Derakshan, 1997 for anxiety; Ehlers & Clark, 2000; Yehuda, 2002 for PTSD).

Ways to measure this risk factor of perceived stress has been varied in the stress research literature. Interestingly, stress research has focused primarily on measuring the perception of stress focusing on the psychosocial effects of stress over the physiological changes of the stress reaction. Therefore, many stress assessments follow in that tradition. Some measurements assess the level of stress a human (or animal) has experienced by inferring how many life events to which the individual has been exposed that are negatively appraised or judged as threatening (Lazarus, 1966). Measurements of stress frequently attempt to infer *perceived stress* by assessing how the individual appraises both the degree of the potential threat and the availability of resources needed to cope or adapt to the threat (Cohen et al., 2016). This appraisal can be influenced by factors including the imminence and duration of the threat, individuals' perceived control of the situation, and individuals' beliefs about themselves and the world (Cohen et al. 2016). Some stress measures have targeted these perceptions of threat and control in specific environments such as the workplace (c.f., Job Control Questionnaire [JCQ], Karasek, Baker, Marxer, Ahlbom, & Theorell, 1981) or in specific relationships such as marriage (Story & Bradbury, 2004).

A different way of quantifying stress is to calculate an individual's amount of actual exposure to stress-inducing events. This objective approach does not capture the individual's perception of stress, but rather the frequency of exposure to events that most other individuals regard as stressful, based on consensus views established in previous

research (e.g., losing a loved one, motor vehicle accidents, and combat experiences) (Monroe & Simons, 1991). This "checklist" strategy of measuring stress has given rise to a wealth of measures known collectively as life events scales. Further research is needed to integrate this objective approach with the perception-based approach described above. An integrated approach would both quantify the number of stressful events an individual had experienced and assess that individual's perception of the stressors, calculating the negative impact of the reported stressors in addition to counting the objective stressors (Sarason, Johnson, & Siegel, 1978).

A third approach to measuring individuals' perception of stress has focused not on individual stress and adaptability towards a specific stressor, but on an individual's self-assessment of coping resources at a single point in time to handle any undefined stressor to be encountered in the near future (Cohen et al., 2016). Cohen's Perceived Stress Scale captures this overarching balance of measuring stress and adaptive coping resources in the present moment (Cohen, Kamarck, & Mermelstein, 1983; Cohen et al., 2016). According to this scale, individuals who have fewer coping resources than sources of stress will report higher ratings of perceived stress than individuals with the inverse proportions. Other measures have adapted the scenario-specific measures mentioned above (e.g., JCQ) to examine the effects of routine stressors in activities of daily life. These measures have been validated as better predictors of health and psychological outcomes than life event scales, as it is the appraisal of stress levels—not the setting or frequency of events—that determines these health outcomes (Cohen et al. 2016; Cohen & Hoberman, 1983; Lazarus, 1966).

In addition, all of the above discussed measures of perceived stress focus on assessing stress for a single point in time or a change in a defined time frame (e.g., past week, past month, or past year). Yet, the effect of chronic stress appraisal and allostatic

load is considered to be cumulative over one's lifetime (Holmes & Rahe, 1967). Longitudinal studies of perceived stress have primarily utilized repeated measurements. Studies have assessed an individual's perception of recent stressors and current coping resources (e.g., using the Perceived Stress Scale) at multiple time points across study duration (Wood, Maltby, Gillett, Linley, & Joseph, 2008; Golden-Kreutz, Browne, Frierson, & Andersen, 2004). Additionally, the stressful life events checklist methodology can be implemented in longitudinal studies with repeated assessment of exposure to these stressful events (Lee, Goudarzi, Baldwin, Rosenfield, & Telch, 2011; Lancaster, Cobb, Lee, & Telch, 2016; Peterson, Duncan, & Canady, 2009; Magnus, Diener, Fujita, & Pavot, 1993). The perception of stressors and frequency of their occurrence are both helpful to conceptualize in a risk analysis for psychopathology.

STRESS BUFFERING FACTORS

If encountering stress (i.e., perceiving threat to one's coping resources to maintain homeostasis) is inherent in daily life and the cascading HPA-controlled physiological reaction inevitable, how can an individual protect oneself from the physical and psychological risks associated with chronic stress exposure? Returning to the cognitive-behavioral theoretical foundations of stress, if the negatively appraised event cannot be resolved, assuaged, or terminated, then individuals rely on factors to enhance perceived coping reserves. Research has identified several factors that can boost our perception of coping resources. These stress-buffering factors lessen the risk of chronic HPA activation and protect individuals against the development of disease and psychopathology.

Social Support

Social support is defined as both tangible and emotional support from others (Cohen & McKay, 1984). Social support can reduce the level of perceived threat that individuals experience, thereby protecting those individuals against the deleterious effects of traumatic stress by increasing their ability to cope successfully with stressful events (Viswesvaran, Sanchez, & Fisher, 1999; Orsillo & Batten, 2005). Following the same findings from the stress methodology research, the *appraisal* of an individual's social support typically had a more powerful effect on the predicted health and psychological outcomes than did an objective quantifiable measure of one's tangible and emotional support (Cohen & McKay, 1984; Langford, Bowsher, Maloney, & Lillis, 1997). The individual's perception of social support was a more powerful predictor than outsiders' objective ratings of social support of the efficacy of social support to palliate (buffer) the harmful effects of chronic stress (Uchino, 2006).

Social support has been shown to act as a powerful buffer to the negative effects of chronic stress on mental health (Cohen & Wills, 1985). Chronically stressed individuals with high levels of social support report fewer symptoms of depression and anxiety compared to their counterparts with low social support (Cohen, 2004; Cohen & Wills, 1985). Two meta-analyses investigating the association of social support with PTSD found that, across 11 studies, social support was negatively associated with PTSD (r = -0.40 in Brewin, Andrews, and Valentine, 2000; r = -0.28 for Ozer, Best, Lipsey, and Weiss, 2003).

Not only is social support a powerful remedy against the immediate effects of stress, but its stress-buffering effects can continue over the long term. In a longitudinal study of the effect of social support, individuals with high social support at baseline were not at risk of increased anxiety or depression symptoms ten years later, even after controlling for exposure to negative life events; individuals with low social support,

however, were at risk for psychopathology as a function of the number of negative life events they had experienced (Dalgard, Bjork, & Tambs, 1995). Higher levels of social support are also associated with lower levels of negative affect (Brummett et al., 2006) and lower levels of future job burnout (Halbesleben, 2006).

Resiliency

Resiliency is defined as maintaining physical and/or psychological health under stress (Liebenberg & Ungar, 2009; Masten, 2001) and the "ability to bounce back or recover from stress, to adapt to stressful circumstances, to not become ill despite significant adversity, and to function above the norm in spite of stress or adversity" (Smith et al., 2008, p. 194; Carver, 1998; Tusaie & Dyer, 2004). This 'bounce back' ability can be understood as a return to healthy levels of psychological and physiological functioning (Gayton & Lovell, 2011; Luthar, Cushing, Glantz & Johnson, 1999; Smith et al., 2008). Resiliency is also conceptualized an individual's ability to maintain healthy levels of psychological and physiological functioning in the face of a potentially traumatic event (Bonanno, Galea, Bucciarelli, & Vlahov, 2007; Bonanno 2004). Across the varying conceptualizations of resiliency, there is agreement in the supporting literature that highly resilient individuals show faster psychological and emotional recovery from stress (Gillespie, Chaboyer, Willis & Grimbeek, 2007).

Resiliency studies have been found to buffer the risk association between stress and psychopathology. A large body of research has examined relationships between resiliency and stress-induced mood and anxiety disorders (Luthar, Cicchetti, & Becker, 2000; Garmezy, Masten, & Tellegen, 1984; Masten & Coatsworth, 1998). Psychosocial factors that influence stress resiliency and protect against depression include positive emotions and optimism, humor, cognitive flexibility, acceptance, altruism, spirituality, and social

support (Southwick, Vythilingam, & Charney, 2005). Psychologically resilient individuals view major stressors as challenges to be accepted, and this cognitive appraisal decreases the risk for presence of depression and anxiety symptoms (Bonanno, 2004; Bonanno, Kennedy, Galatzer-Levy, Lude, & Elfström, 2012).

While much of the research on resiliency to date has been done in populations with exposure to prototypical traumatic events such as natural disasters, traumatic injury, combat exposure, and abuse (Bonanno et al., 2012), less information on the influence of resiliency in the stress response in the face of long-term, chronic stress. However, emotional regulation has been identified as a putative mechanism in influencing an individual's stress reaction (Ong, Bergeman, Bisconti, & Wallace, 2006). More resilient individuals show less emotional dysregulation in the face of stress and traumatic events due to the cognitive appraisal of their own adaptiveness and future recovery ('bounce back') from the stressor. Greater emotional dysregulation predicts greater risk of emergence of depression, anxiety, and PTSD, indicating that less resilient individuals are placed at higher risk for development of these affective psychopathologies than their highly resilient peers. The practice of strengthening emotional regulation is currently utilized by a wide range of evidence-based psychotherapeutic interventions for psychopathology, such as cognitive processing therapy (CPT) (Resick & Schnicke, 1992) and skills training in affective and interpersonal regulation (STAIR) (Cloitre, Koenen, Cohen, & Han, 2002). These intervention approaches share a key goal in minimizing clinical symptomology through building and reinforcement an individual's perceived social support and resiliency.

Given the established research literature of perceived social support and resiliency to buffer the pathologic association of stress with psychopathology, this dissertation follows in the tradition of stress assessment and interventions to quantify these factors and assess their predictive power in reducing an individual's risk of depression, anxiety, and

PTSD symptomology and other disorder-related behaviors (such as, poor sleep and increased substance use).

DIATHESIS-STRESS MODEL FOR STRESS-LINKED PSYCHOPATHOLOGY

One common method of theorizing the mechanisms for the stress-related risk and protective factors is a conceptualization of the factors in play in a frame work containing "diatheses" and environmental stressors. Because not every individual who encounters chronic stress develops downstream clinical affective disorders, there must exist intra-individual differences that affect a person's ability to adapt to life's stressors which regulate physical and psychological functioning (Selye, 1936; Taylor & Sirois, 1995).

When an individual's adaptation to stress is dysregulated or overwhelmed, stress is associated with higher levels of psychological and emotional distress (Cohen & Wills, 1985); however, Meehl (1962) was one of the first researchers to clarify that not all individuals develop emotional distress and psychological disturbance in the face of high stress. He hypothesized that vulnerabilities to stress-linked disease within a population are due to these individual differences – termed diatheses – that may be considered risk factors for developing stress-linked psychopathology (Meehl, 1962). In the 1960s, researchers conceptualized this phenomenon further in studies of schizophrenia (Bleuler, 1963; Walker & Diforio, 1997). They demonstrated that an individual could be assessed to either have a presence or absence of a risk factor (diathesis) that places the individual at a greater vulnerability for development of emotional disturbance. Commonly investigated diatheses include characteristics of temperament (Tackett et al., 2014; Zuckerman, 1999), gender (McHenry et al., 2014), and genetic polymorphisms due to inherent population variation (Caspi et al., 2003).

The second component of the diathesis-stress model is the individual stress response – physiological and psychological - to a stressor which has been discussed at length in the above sections. However, individuals do not have identical reactions to the same environmental stressor. In fact, some individuals may be entirely emotionally unperturbed by the environmental stressors, while others may develop emotional dysregulation and psychiatric distress, possibly to a diagnosable severity (Dohrenwend, 2000; Kessler, Chin, Demler, Merikangas, & Walters, 2005; Yehuda & LeDoux, 2007). What is the difference between these variable reactions to the same environmental stressors? Researchers conceptualized that differentiating factors in the stress reaction were due to diatheses. Therefore, the presence of the diathesis alone, or of external stressors alone, is not sufficient for the development of psychological disturbance; rather, the two must occur in combination with one another. Monroe and Simon (1991) define the diathesis-stress framework (see Figure 3) as follows: "stress activates a diathesis, transforming the potential of predisposition into the presence of psychopathology" (p. 406).

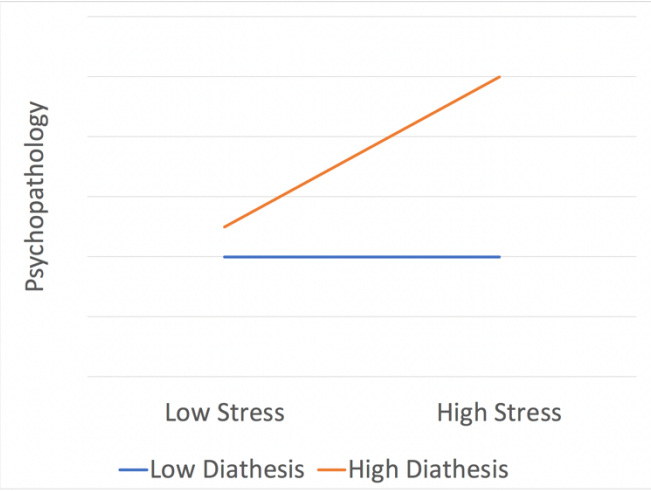

Figure 3: Diathesis-stress model of stress-linked psychopathology.

Over the latter half of the 20th century, researchers have used diathesis-stress framed models to better understand a variety of psychopathologies including depression (Caspi et al., 2003; Monroe & Simons, 1991), personality disorders (Beck, Davis, & Freeman, 2015; Fruyt & De Clercq, 2014), schizophrenia (Walker & Diforio, 1997), externalizing disorders (Rioux et al, 2016), psychopathy (Tielbeek et al., 2016), substance use (Goldstein, Buchanan, Abela, & Seligman, 2000), and sleep disturbance (Drake, Pillai, & Roth, 2014).

In the current study, high levels of baseline cortisol are considered a risky diathesis for clinical symptoms of affective mental illness such as depression and anxiety (Holsboer, 2000; Burke et al., 2005; Nemeroff & Vale, 2005; Heuser et al., 1994). Lower baseline cortisol levels have been shown to confer risk associated with the psychiatric illness in some studies (Jezova, et al., 2004; Yehuda, 2001). The cortisol-PTSD literature presents a mixed picture, with some support for a positive association between hypercortisolism and PTSD (Yehuda, 2005; Violanti et al., 2007; Stoppelbein, Greening, & Fite, 2012) and some support for a positive association between hypocortisolism and PTSD (Meewisse et al., 2008; Lauc et al., 2004; Yehuda, 2001). With regard to testosterone and psychopathology, evidence supports low baseline testosterone as a risky diathesis for depression and anxiety, with higher baseline testosterone showing protective effects (Booth et al., 1999; McHenry et al., 2014; Bitran et al., 1993). Low levels of social support are also risky diatheses for clinical psychopathology (Cohen, 2004; Cohen & Wills, 1985; Dalgard et al., 1995) as is low resiliency (Bonanno, 2004; Bonanno et al., 2012; Luther et al., 2000).

These four diatheses (i.e., high cortisol levels, low testosterone levels, low perceived social support, and low perceived resiliency) are conceptualized to predict increased levels of psychopathology in a longitudinal study of first responders. In addition

to single-hormone models, the interactive effects of cortisol and testosterone will serve as dual diatheses for prediction of psychopathology.

STRESS AND FIRST RESPONDERS

The research discussed above establishes the theoretical conceptualizations and research findings for physiological and psychological diatheses in the stress response with a plethora of evidence that certain neurobiological and psychosocial factors can serve as buffers against negative stress outcomes such as stress-linked mental illness. This approach to understanding stress enables clinicians and scientists to better understand the possibilities of more effective treatment for all individuals, not just for first responders. However, this dissertation study focuses on first responders as a critical population because the extremely stressful nature of their occupation provides a rare opportunity to study the dynamics involved in the association between persistent bouts of extreme stress and psychopathology. It is expected that stress-buffering neurobiological and psychosocial factors will play out strongly in this population, and that endocrine factors of the diathesis-stress model are interacting within the disease framework. The remaining literature review will delve into the research to date on the measurement of stress followed by the measurement of the four proposed diatheses – cortisol, testosterone, perceived social support, and resiliency – within the first responder population specifically.

Measuring Stressors Using Critical Incidents

Traumatic situations experienced by first responders are known as "critical incidents" (Kleim & Westphal, 2011; Marmar et al., 2006). Critical incidents may include but are not limited to violent accidents, injury or death of adults or children, fires, mass

casualties, and suicides (Beaton, Murphy, Johnson, Pike, & Corneil, 1999). In the latest version of the *Diagnostic and Statistical Manual of Mental Disorders, 5th Edition* (DSM-5), the repeated witnessing of threats of violence, injury and death while on the job as a first responder was added to the criteria of "exposure to actual or threatened death, serious injury, or… violence" for PTSD (American Psychiatric Association, 2013, p. 271). Compounding these already-stressful events, first responders' experiences at the scene often include threats to their own lives and safety (e.g., fire, violence, and accidents). Rutkow, Gable, and Links (2011) describe this profession as fraught with "long hours under stressful conditions, witnessing the human harms, physical destruction, and psychological devastation that can accompany disasters" (p. 56). Under these distressing job conditions, first responders are expected to perform their duties with the highest quality of care.

A first responder is highly likely to be exposed to one or more critical incidents at work deemed to be stressful. According to a 2003 study, the top five most stressfully rated critical incidents involved dead children, medical emergencies, severe accidents, violent or threating environments, and suicide attempts (van der Ploeg & Kleber, 2003). In a study of firefighters, Sattler, Boyd, and Kirsch (2014) reported that 94% of participants were exposed to critical incidents in their career, and the most reported traumatic critical incidents were fatalities, injury or death of a child, violent situations, providing medical response to friends or family, body retrieval, and the risk of injury or death to the firefighter. Studies of paramedics have confirmed exposure to at least one critical incident, including violent environment (93%), fatality (91%), death of a patient (85%), death of a child (85%), real or threatened injury or harm to the paramedic (70%), near-death experience (56%), and a fellow responder's death in the line of duty (28%) (Regehr et al., 2002).

Another challenging component of first responders' job is the empathetic experience generated by witnessing others' traumas (Alexander & Klein, 2001). Studies use the terms "secondary trauma" or "vicarious traumatization" to describe the emergence of posttraumatic stress symptoms in first responders who work with trauma victims (Regehr et al., 2002, p. 505). First responders who arrive at scenes of violence, destruction, or death are more likely to experience high levels of traumatic stress compared to those who do not experience such on-the-job scenes (Stinchcomb, 2004). As noted by Sattler et al. (2014) most (greater than 90% of) first responders in their career will sadly encounter these traumatic scenes.

The magnitude of emotional distress in first responders is greater than that of employees in other health professions except emergency room nurses (van der Ploeg & Kleber, 2003). Van der Ploeg and Kleber (2003) reported that 85% of ambulance personnel had experienced a critical incident that they described as personally distressing within the last five years, and 66% had experienced a distressing critical incident within the last year. Another study found that 82% of ambulance personnel reported experiencing a disturbing critical incident within the last six months (Alexander & Klein, 1991). Regehr and colleagues (2002) found that 82% of paramedics reported being overwhelmed or unsettled by a critical incident, with 78% reporting distress both during and after the event. Paramedics described the most distressing or unsettling critical incidents as those that involved suicide or violence against children (Regehr et al., 2002). Compounding these secondary traumas is the fact that 70% of first responders reported that they do not feel they have sufficient time to emotionally process and recover between traumatic events (Alexander & Klein, 2001). Various research in non-first responder professions estimated that 75% of emergency room physicians reported one or more trauma exposures within the last six months (Somville, Gucht, & Maes, 2016). Only a subset of hospital workers –

emergency room nurses – had a comparable trauma exposure rate (87%) of at least one traumatic event in the last six months (Adriaenssens, Gucht, & Maes, 2012). While the field reports high levels of secondary traumatic stress (STS) in social workers (87%) and therapists (>50%), the risk of the pathologic effects of trauma are minimized by the indirect exposure to the trauma (e.g., during psychotherapeutic treatment) rather than direct involvement in the medical trauma or immediate aftereffects (Bell, Kulkarni, & Dalton, 2003; Bride, 2007). Due to these factors, first responders are more likely to report post-traumatic stress symptoms than are the majority of health care workers (Beaton & Murphy, 1995).

On top of high frequency of exposure to traumatic critical incidents on the job, first responders generally report higher levels of chronic work-related stress relative to the general population (van der Ploeg & Kleber, 2003). Compared to people who work in other health professions, paramedics report greater stress due to lack of job autonomy, physical demands, lack of support from peers and supervisors, poor communication, and non-competitive financial rewards (van der Ploeg & Kleber, 2003). Higher stress was associated with emotional exhaustion, lower levels of job satisfaction, and higher posttraumatic symptoms (Alexander & Klein, 2001). In a 2003 study of first responders, 8% of participants met the criteria for job burnout, and 10% were considered at risk for taking sick leave and disability (van der Ploeg & Kleber, 2003). Other prevalence studies for first responders found percentages as high as 50% for personal burnout (Stassen, Van Nugteren, & Stein, 2013) . The strongest predictors of burnout and low job satisfaction in paramedics are high levels of workplace environment stressors, including lack of support from colleagues or supervisors, and poor communication (van der Ploeg & Kleber, 2003). This at least 50% prevalence rate for burnout is commiserate with work-related burnout of US physicians (Shanafelt, et al., 2014) and higher than burnout reported in a recent

professional survey for US physicians, which reported 38% overall physician burnout and 44% burnout amongst emergency medicine physicians (Kane, 2019; Frellick, 2019).

Off-the-job stress also contributes to the clinical picture of stress responses in first responders as their experience of stressors does not turn off when they clock off a shift. Studies assessing psychological markers of stress (self-reports of perceived stress) and physiological markers of stress (salivary cortisol) in paramedics revealed no significant differences during 24-hour work shift days versus days off, suggesting that stress in first responders should be assessed both during work and during off periods for a fuller clinical understanding (Aasa, Kalezic, Lyskov, Ängquist, & Barnekow-Bergkvist, 2006).

PREVALENCE OF PSYCHOPATHOLOGY AMONG FIRST RESPONDERS

Stress-linked psychopathology is present in paramedic populations at a higher prevalence than in the general population (Morganstein, Benedek, & Ursano, 2016; Gayton & Lovell, 2012; Beaton, 2006; Alexander & Klein, 2001). In the general US adult population, the 12-month prevalence estimates are 26.2% for any psychiatric disorder, 18.1% for anxiety disorders, 9.5% for mood disorders (6.6% for major depression), and 3.5% for PTSD (Kessler et al., 2005; Kessler et al., 2003). Substance use disorders in the general US adult population had a 2-3% 12-month prevalence for illicit drugs and as high as 12% 12-month prevalence for alcohol use (Merikangas & McClair, 2012).

In contrast, the most frequently reported symptoms of psychopathology in first responders are posttraumatic stress disorder (PTSD) (Cone et al., 2015; Haugen et al., 2012), major depressive disorder (Benedek, Fullerton, & Ursano, 2007; Fullerton, Ursano, & Wang, 2004), and drug and alcohol-related disorders (Cross & Ashley, 2004). Kleim and Westphal (2011) estimated that the point prevalence of PTSD, depression, and mental

health diagnoses in first responders ranges from 8-32% for PTSD, 16-26% for depression, and 27% for any mental health disorders. While first responder prevalence for any psychiatric diagnosis is similar to the general population, prevalence for depression and anxiety are disproportionally represented within the group as these rates are exorbitantly higher than rates are much high than the 6.6 and 3.8% 12-month prevalence of the general population, respectively. Furthermore, unpublished data assessing for presence of clinical symptoms of mental disorders in first responders (recruited from the same population in which this dissertation study is sampled) showed that first responders reported greater levels of trauma exposure, PTSD, and depression compared to the lifetime prevalence of trauma exposure and the annual prevalence rates of depression and PTSD in the general population (Rice, 2014). These findings offer a snapshot of the mental health status of local first responders at only one point in time; thus, they do not advance our understanding of how mental health symptoms change over the long term within this profession. Clearly, then, longitudinal research on first responders is needed to assess trends of psychopathology across time in this critically important population.

Prevalence of PTSD among First Responders

First responders report higher levels of PTSD than are reported in the general population, presumably due in part to greater trauma exposure from the job (Kleim & Westphal, 2011). In a review of PTSD treatment studies for first responders, Haugen et al. (2012) report that out of a conservatively estimated 1.5 million first responders, "nationwide, there may well be a quarter of a million first responders impaired by symptoms of PTSD for whom effective interventions would be both compassionate and utilitarian" (p. 371); they add that this statistic likely underestimates the true prevalence of first responders who could benefit from treatment interventions. Similar to Kleim and

Westphal (2011) reporting an upper bound of PTSD point prevalence of 32%, another study revealed that the PTSD prevalence rate for paramedics may be as high as 37% (Clohessy & Elhers, 1999). In a study of Swedish paramedics, 15% were diagnosed with probable PTSD (Jonsson, Segesten, & Mattson, 2003); in a 2004 study, 22% of British first responders were diagnosed with probable PTSD (Bennett, Williams, Page, Hood, & Woollard, 2004). Studies of paramedics have found higher prevalence of PTSD than studies of police and firefighters (Berger et al., 2012). The prevalence of PTSD in 9/11 first responders over the 10 years following the disaster was similar, ranging from 10% to 20% (Bromet et al., 2016; Cone et al., 2015). Another study of 9/11 first responders found that PTSD symptoms were associated with the interaction between 9/11 acute trauma exposure and chronic stressful life events (Zvolensky et al., 2015). Researchers have also found that the risk of PTSD is not related only to the quantity of stress exposures, but also to the diversity of stress exposures (Geronazzo-Alman et al., 2016). These studies reinforce the view that first responders are at greater risk of PTSD based on higher quantity, frequency, and diversity of trauma exposure they experience.

Prevalence of Depression and Anxiety among First Responders

Perceived chronic stress, disrupted sleep due to shift work, and critical incidents on the job are associated with higher depression scores in the first responder population (Benedek et al., 2007). As with PTSD, depression prevalence rates are also variable within the first responder population. Regehr and colleagues (2002) found that 19.5% of firefighters reported moderate depression, and 3% of firefighters reported severe depression. In a study of Japanese firefighters involved in fire and emergency service work, 21% were reported to suffer from depression (Saijto, Ueno, & Hashimoto, 2008). A small (n=60) pilot study of paramedics in Australia reported mild depression in 27% and

moderate depression in 10% of the sample (Sofianopoulos, Williams, Archer, & Thompson, 2011). The above prevalence rates of depression in these epidemiological studies are higher for paramedics than for the general population at 6.6% for 12-month prevalence (Kessler et al., 2005).

Prevalence estimates for anxiety disorders in first responders are lacking when compared to the numerous studies of PTSD and depression (c.f. Kleim & Westphal, 2011). An earlier dissertation study studying first responders nationally found approximately 12% point prevalence of anxiety using optional screening surveys sent to first responders during the recertification paperwork process (Bentley, 2011). Our pilot data collection showed a point prevalence of 99% trauma exposure but did not explore more general anxiety disorder other than PTSD symptoms using non-PTSD screening surveys (Rice, 2015; Kruse et al., 2013). In a related high-risk field, a study found congruently that trauma nurses reported higher levels of general anxiety than their non-trauma nurse counterparts. These results are suggestive that trauma-focused professions, such as first responders, are at relative risk for anxiety diagnoses at or greater than 18.1% 12-month prevalence seen in the general population (Kessler et al., 2005). This dissertation ensured anxiety screening measures outside of PTSD-specific inventories in order to broaden our understanding of point prevalence and 6-month prevalence of first responders.

Prevalence of Alcohol Use and Sleep Disturbance among First Responders

The prevalence rates of problematic alcohol use in first responder population does not statistically differ from the rates general populations for men, but that is not the case for women. The first responder population has been found to have a 7.8% prevalence of lifetime alcohol use disorder; while this is comparable to males' rates of alcohol use disorder in the general population, female first responders "were 1.6 times more likely than

women in the general population to have had greater than 14 drinks in the past week" demonstrating a much higher point-in-time prevalence for alcohol use disorder than their non-first responder female counterparts (Marmar et al., 2006, p. 9).

As in the general population, increased substance use is comorbid with stress-linked psychopathology among first responders. In this population, increased substance use is considered a mental health consequence of exposure to natural and man-made emergencies (Alexander & Klein, 2009; Benedek et al., 2007). Increased substance use following traumatic events has been described as a readily-utilized short-term coping strategy for first responders (Regehr et al., 2002). In support of this perspective, the percentage of first responders reporting alcohol use increased from 1.2% to 11.6% following exposure to critical incidents (Regehr et al., 2002). In another troubling finding, some first responders report that substance use after a critical incident reduced their ability to cope with future stressors and increased their habits of problematic drinking (Regehr et al., 2002). For example, one former first responder summarized alcohol's deleterious effects as a coping mechanism for himself and his environment using the following vivid imagery: "I just basically burned out and fell into a pot of booze. Then I quit because it was killing me, killing my family, killing my work" (Regehr et al., 2002, p. 508).

In addition to their increased risk of substance use compared to the general population, first responders also face a higher risk of sleep disturbance. The point prevalence of sleep disturbance in the first responder population has been assessed as high as 72% in samples (Courtney, Francis, & Paxton, 2010; Sterud, Ekeberg, & Hem, 2006). This is in stark contrast to the estimated 20-41% prevalence compiled from meta-analyses of epidemiological studies among the general population (Ohayon, 2011). Survey data from first responder samples show that sleep problems adversely affect energy levels, mental health, and job performance (Sofianopoulos et al., 2011; van der Ploeg & Kleber,

2003). Sleep problems are compounded by the nature of the job. Long work shifts vary from 9-12 hours up to a full 24 hours, and these shifts occur on multiple days of the week, possibly back-to-back (Konig, 2016; Walker, McKune, Ferguson, Pyne, & Rattray, 2016). This grueling work schedule is associated with self-reported fatigue and sleep disturbance (Vila, Samuels, & Wesensten, 2017; Courtney et al., 2013; Sofianopoulos et al., 2011). While increased stress is positively associated with worsened sleep for any individual (Åkerstedt, 2006; Van Reeth et al., 2000), exposure to more critical incidents in the field increases a first responder's risk for sleep disturbance (Regehr et al., 2002; Everly, Flannery, & Mitchell, 2000). Additionally, the majority of first responders (88%) also perceive that fatigue and sleep are likely affecting the quality of their work performance in the field (Sofianopoulos et al., 2011).

Sleep problems, like substance use problems, can be both antecedent to, and symptomatic for stress-linked psychopathology. The DSM-5 includes fatigue and sleep disturbance as symptomatic criteria of major depressive disorder and PTSD (American Psychiatric Association, 2013). The most common PTSD symptoms reported by first responders include sleep disturbance and irritability (Cone et al., 2015; van der Ploeg & Kleber, 2003). In a recent study, paramedics who had experienced PTSD or depression episodes over a two-year period reported significantly worse sleep relative to those who did not experience a mental health disorder episode (Wild et al., 2016). The significant impact of sleep disturbance demonstrates that sleep quality should be evaluated within the context of stress and mental health research in first responders and will be assessed in this dissertation.

Maintenance of Psychopathology among First Responders

Of the few studies that have examined mental health in first responders, most have been cross-sectional (Kleim & Westphal, 2011; Alexander & Klein, 2001). The few longitudinal studies tracking emergency responders' responses to large-scale disasters, such as earthquakes or the World Trade Center terrorist attack, show the continued maintenance of psychiatric distress and mental illness for many years following the critical incident (Bromet et al., 2016; Zvolensky et al., 2015; Marmar et al., 2006; Fullerton et al., 2004). For example, studies following first responders to the World Trade Center disaster have reported long-lasting PTSD and depression prevalence: 9.7% current PTSD prevalence in responders 11-13 years later (Bromet et al., 2016), 7% prevalence of possible PTSD up to 12 years later (Yip et al., 2015), and 9.3% prevalence of PTSD and 7% prevalence of depression in rescue and recovery workers 9 years following the attack (Wisnivesky et al., 2011). Chronic symptoms of PTSD (including hyperarousal) and emotional distress in first responders were present almost 4 years after the Loma Prieta Bay Area Earthquake (Marmar et al., 2006), and the rate of PTSD at six months following the Wen Chuan earthquake was 6.5% (Wang et al., 2011). These findings highlight how just one large-scale critical incident can have deleterious and long-lasting effects on the mental health of first responders.

Another factor that may increase long-term presence of psychopathology in a first responder population is the frequency of critical incidents to which the first responder is exposed. There is evidence that the number of critical incidents is associated with the duration of post-traumatic symptoms, fatigue, and burnout in paramedics (van der Ploeg & Kleber, 2003). Findings from military samples highlight the importance of considering the number of potentially traumatic stressors as a factor that may predict the magnitude and duration of psychopathology (Lancaster et al., 2016; Lee et al., 2011; Telch, Harrington,

Smits, & Powers, 2011). Studies that followed in-theater combat soldiers over deployment *(M*=14 months) have indicated maintenance of psychopathology due to the persistence of the stress-linked variables (emotional regulation, testosterone, and cortisol) reinforcing the importance of prospective, longitudinal designs (Lee et al, 2011; Telch et al., 2011, Lancaster et al. 2016, Josephs et al., 2017, Cobb et al. 2018).

STRESS-LINKED HORMONES AND FIRST RESPONDERS

Cortisol Studies among First Responders

Despite the significant body of literature linking HPA axis dysregulation to mental health, there is currently little research on the effects of cortisol on first responders' mental health outcomes. Further research reports that shift work and disrupted sleep—common features of first responder duties—are mediating variables in the positive association between HPA dysregulation leading to higher basal cortisol (Touitou et al., 1990) and sleep disturbance (Niu et al., 2011). Walker et al. (2016) reported that sleep restriction might adversely influence the acute stress-evoked cortisol response that—under normal conditions—would enable first responders to process acute traumatic events in a healthy and adaptive way. The authors also reported that other harsh work environments experienced by first responders, such as heat, smoke, physical exertion, and overtraining, have the potential to dysregulate the stress-evoked cortisol response.

In one of the few studies of cortisol activity in first responders, a study of Dutch paramedics found that cortisol activity—measured by collecting saliva during and after response to a critical incident—showed more dysregulated cortisol recovery (i.e., cortisol levels did not return to pre-incident levels by the follow-up assessment) in paramedics responding to severe or life-threatening emergencies, compared to paramedics responding

to less severe or non-life-threatening emergencies (Sluiter, van der Beek, & Frings-Dresen, 2003). More evidence for the adverse effect of stress on cortisol activity in first responders comes from LeBlanc et al. (2012), who instructed paramedics to participate in either a low-stress or a high-stress simulated critical incident. Results showed higher cortisol levels and higher accompanying subjective anxiety in paramedics assigned to the high-stress critical incident conditions. Further, LeBlanc and colleagues (2012) found that when performance on the simulated critical incident was evaluated by independent reviewers, paramedics with higher cortisol levels produced lower global ratings of performance, regardless of the objective stress-level of the critical incident. Lower performance was characterized by poorer organization, communication, and interpersonal skills (LeBlanc et al., 2012; LeBlanc et al., 2011). Taken together, these findings highlight that cortisol is an important biomarker for stress and allostatic load as HPA axis function plays a role in first responder adaptation to stress, mental health, and job performance.

Testosterone Studies among First Responders

Similar to cortisol, testosterone has been implicated in the regulation of acute and chronic stress (Mehta et al., 2015; Josephs, Sellers, Newman, & Mehta, 2006; van Honk et al., 1999). Further, low testosterone levels have been associated with increased risk of depression and anxiety (McHenry et al., 2014). In first responders, however, the literature on testosterone and stress on mental health is limited. In the few studies located, testosterone levels were found to moderate the effect of the cognitive appraisal of fear on firefighting performance and conscientiousness in an emergency situation (such as a fire), with higher testosterone levels supporting better performance and higher conscientiousness (Fannin & Dabbs, 2003). However, a separate first responder-specific study found no association between prenatal testosterone levels (measured using the second digit-to-fourth

digit of the hand (2D:4D) ratio) and firefighter performance (Voracek, Pum, & Dressler, 2009); however, this study did not measure testosterone directly, but rather used the digit ratio as a proxy measurement to for testosterone levels as the 2D:4D is used in the endocrine literature as a non-invasive procedure to estimate the sexually dimorphic levels of androgen exposure in fetal development that carries into adulthood (Manning, Bundred, Newton, & Flanagan, 2003; Bailey & Hurd, 2005).

Dual Hormone Effects of Cortisol and Testosterone among First Responders

An extensive literature review revealed no studies (to the author's knowledge) investigating the joint, stress-moderating effects of testosterone on the association between stress (cortisol levels and perceived stress levels) and psychopathology in a population of paramedics or other first responders. As mentioned earlier in this review, U.S. soldiers whose testosterone was highly reactivity to a pre-deployment acute stressor (a 35% CO_2 stressor) did not show war zone stress-linked PTSD symptomatology, compared to their low testosterone reactivity counterparts (Josephs et al., 2017). Further, high cortisol reactivity was risky for stress-induced depression within the population (Cobb et al., 2019). Additionally, low testosterone individuals with high testosterone reactivity to a threatening stressor were at greater risk for stress-induced depression symptoms. Interestingly no duel hormone effects were found when predicting depression like was found in the PTSD models. However, the separate effects found of each hormone in depression gives strong evidence in including both hormones for future dual hormone investigations (whether interactive effects are expected). Further, similar protective hormonal effects have been reported by Cobb et al. (2018), who reported that lower basal testosterone levels were linked to in-theater, stress-evoked depression symptoms during deployment while those with higher testosterone found a neuroprotective benefit against depression symptoms.

These provocative findings suggest that the interaction of cortisol and testosterone might provide a better understanding of the relationship between stress and psychopathology within the first responder population. This dissertation will be an early study to include both effects of both hormone diatheses into the psychopathology prediction models. Interpretations of these models' factors are hoped to add meaningful information to the first responder research literature.

SOCIAL SUPPORT AND RESILIENCY AMONG FIRST RESPONDERS

Among first responders, social support has been shown to be a robust, negative predictor of PTSD symptoms (Kleim & Westphal, 2011; Sattler et al., 2014; van der Ploeg & Kleber, 2003; Cone et al., 2015). Prati and Pietrantoni (2010) conducted a meta-analysis on the association of perceived social support with mental health among first responders, reporting a medium effect size ($r = 0.27$) of the positive relationship between higher perceived social support and better mental health outcomes. These effects attest to the importance of social support in preserving first responders' mental health. In fact, some have argued that social support, particularly from one's superiors in the profession, may be *the* primary protective factor within a first responder's personal and organizational network (Kleim & Westphal, 2011; Regehr, Hill, & Glancy, 2000; Leffler & Dembert, 1998).

The limited resiliency research to date within the first responder population suggests that paramedics possess a "hardy" or resilient personality (Gayton & Lovell, 2012; Alexander & Klein, 2001). However, few studies have attempted to quantify this resiliency within paramedics. One exception is Gayton and Lovell (2012), who found that the higher the first responder's resiliency was significantly associated with better health and well-being than compared to their first responder peers with lower resiliency ratings. Higher

resiliency was also associated with greater life satisfaction. Based on these findings, measuring resiliency in first responders is indicated to better understand the relationship between acute and chronic stressors and mental health. Including this factor in prediction models of stress-linked psychopathology will give researchers more information on the appropriate measurements of resiliency as a 'hardiness' trait amongst first responders. Finally, research suggests that developing interventions to target resiliency and mental health could have bidirectional effects; raising resiliency levels may likely improve mental health in the first responder as well as general population, while targeting mental health symptoms may lead to increased resiliency.

Chapter 2: The Present Study

Evidence from studies on first responders illustrates that these individuals are distinct from other populations in that they endure repeated exposures to stressors, including potentially traumatic stressors, in addition to the negative effects of chronic work stress. A vicious cycle of stress exposure and development of psychopathology has been investigated in first responders (Kleim & Westphal, 2011; Benedek, et al., 2007; Cross & Ashley, 2004; Clohessy & Elhers, 1999; Jonsson, et al., 2003), although a lack of longitudinal studies limits the confidence of scientific generalization of findings (c.f. Haugen et al., 2012). To date, it is unclear to what degree stressors interact with first responders' individual diatheses (hormone levels, perceived social support, resiliency ratings) to predict risk for future presence of symptoms of PTSD, depression, anxiety, substance use disorders, and sleep disturbance.

In this dissertation, a sample of first responders are investigated to explore the predictive accuracy of a diathesis-stress framework of stress-linked psychopathology. Within the model framework, several diatheses (specifically, baseline basal cortisol, baseline basal testosterone, baseline perceived social support, and baseline resiliency) are hypothesized to moderate the effects of stress on the emergence of PTSD symptoms, depression symptoms, anxiety symptoms, alcohol use, and sleep disturbance over a 6-month period. As data were collected over a 6-month time frame tracking for changes in clinical outcomes within individual first responders, this study is prospective or longitudinal in theoretical study design rather than cross-sectional. However, time is not modeled as a specific variable into the diathesis-stress models, but rather baseline predictors are used to predict psychopathology at 3-month and 6-month follow-up. Given the availability of longitudinal data to track stress-related clinical symptoms over time for

these first responders, diatheses and stress levels at baseline levels are used to develop prediction models for clinical symptomatology or related clinical health-behaviors in the future (i.e., at three months and six months following baseline). The choice of 3-month and 6-month follow-ups is supported by findings of Alexander and Klein (2011) that paramedics who reported a critical incident within the last six months were most likely to also report general psychopathology. Further, Haugen et al. (2012) argue that there exists a critical and unfulfilled need for studies with longitudinal designs to explore the health and well-being of first responders for periods of at least six months.

The current study is novel in that it investigates the predictive effects of dual hormones, stress, social support, and resiliency on stress-linked mental health symptoms in a population of first responders. A better understanding of the significant temporal effects of a stressor to predicting manifestation of future symptoms could provide clinicians with insight into a possible "sensitive period" following significant trauma exposure to influence mental health symptomology of its service members and introduce stress-management behavioral interventions. This study assumes that all participants at baseline have differing levels of stress levels as well as baseline levels of clinical health symptoms. To account for these individual differences, this study will focus on intra-individual scores in clinical symptoms as clinical gold-standard stress management techniques and psychotherapeutic interventions are applicable for reducing symptoms in individuals no matter starting symptom severity or duration. Additionally, baseline clinical scores of outcome variables will be modeled as a predictor across all regression models to control for these inter-individual differences in stress-linked mental health.

STUDY GOAL

The goal of this dissertation study was to utilize a diathesis-stress framework to explore the role played by endocrine risk factors in: a) PTSD (Josephs et al., 2017); b) depression (Cobb et al., 2018); c) anxiety (Jezova et al., 2004); d) substance abuse (Walther, Rice, Kufert, & Elhert, 2016); and e) poor sleep quality (Riemann et al., 2015) in a sample of first responders. In this study, two of the diathesis candidates were represented by the dual effects of testosterone and cortisol as described in the dual hormone hypothesis (Mehta & Josephs, 2010). Both hormones have been implicated in dysregulated stress responses (Miller et al., 2007; McEwen, 2004; Roy et al., 2003) and greater risk of increased symptoms of psychopathology (McHenry et al., 2014, Burke et al., 2005; Jezova et al., 2004). Here I predict a joint effect of these two hormones on stress-linked psychopathologies, in which high levels of testosterone are capable of suppressing the pathogenic effects of cortisol on the development of mental health symptoms. Studies looking at the interaction between basal cortisol and testosterone are mixed, but the majority of these studies show high cortisol to be pathogenic – i.e., testosterone's protective effect was blocked; therefore, high cortisol and low testosterone are the pathogenic dual profiles. (Mehta & Prasad, 2015). This same literature review shows that there is the reverse effect (low cortisol is pathogenic) in several studies. The other two diathesis candidates were perceived social support and psychological resiliency, both of which were expected to serve as protective stress buffers acting upon the stress response. These were predicted to show significant effects in attenuating any pathogenic outcomes of the endocrine effects on stress-linked psychopathology.

This dissertation study adopted a prospective, longitudinal design, in which diatheses (testosterone and cortisol concentrations, perceived social support, and resiliency) as well as perceived stress ratings were collected at baseline while

psychopathology clinical symptom inventories were assessed at baseline, but also at 3-month and 6-month follow-up. These diatheses were investigated in ordinary least squares (OLS) regression models to assess associations with changes in PTSD, depression, anxiety, alcohol use, and sleep disturbance symptoms across 3-month and 6-month follow-ups.

Each hypothesis listed below tested for the effect of baseline diatheses in moderating baseline perceived stress for each of these five clinical outcomes: PTSD, depression, anxiety, alcohol use, and sleep disturbance at three months and six months from baseline in a first responder sample. To clarify all data prediction models will use only baseline perceived stress variables from responders rather than using multiple perceived stress ratings capture throughout the study time frame.

STUDY AIMS

Aim 1: Moderating effects of single hormonal diathesis to predict stress-linked psychopathology at three and six months from baseline.

1A. Investigate the moderation of cortisol on the positive association between high stress and risk of psychopathology at 3-month and 6-month follow-up.

1B. Investigate moderation of testosterone on the positive association between high stress and risk of psychopathology at 3-month and 6-month follow-up.

Hypotheses for Aim 1

1A. High baseline cortisol will interact with high levels of stress to predict increased clinical symptoms at three months following baseline. It is expected that this interaction of cortisol and high stress will continue to be impactful at six months following baseline.

1B. High baseline testosterone will interact with high levels of stress to predict decreased clinical symptoms at three months following baseline, and its effects are expected to persist at six months following baseline.

Aim 2: Moderating effects of dual hormonal diatheses to predict stress-linked psychopathology at three and six months from baseline.

Investigate the dual moderation of cortisol and testosterone on the positive association between high stress and risk of psychopathology at 3-month and 6-month follow-up.

Hypotheses for Aim 2

2. High baseline testosterone will blunt the pathogenic effect of high cortisol on stress-linked clinical symptoms at three months following baseline. Specifically, among first responders with high cortisol levels, those with also high testosterone levels will report lower symptoms than their low-testosterone peers due to the stress-buffering effects of testosterone. It is expected that this joint moderation will persist at six months following baseline.

Aim 3: Moderating effects of perceived social support in combination with a single hormone diathesis to predict stress-linked psychopathology at three and six months from baseline.

3A. Investigate the moderation of baseline perceived social support and cortisol on the positive association between high stress and risk of psychopathology at 3-month and 6-month follow-up.

3B. Investigate the moderation of baseline perceived social support and testosterone on the positive association between high stress levels and risk of psychopathology at 3-month and 6-month follow-up.

Hypotheses for Aim 3

3A. High levels of social support will blunt the pathogenic effect of high cortisol on stress-linked clinical symptoms at three months following baseline. Specifically, among first responders with high cortisol levels, those with high social support levels will have lower symptoms than their low social support peers due to the stress-buffering of social support. It is expected that this joint moderation will persist at six months following baseline.

3B. High levels of social support will interact with high levels of testosterone to predict decreased symptoms at three months following baseline. Specifically, among first responders with high testosterone levels, those with high social support levels will predict even fewer symptoms than those their low social support peers due to social support's stress buffering. This effect will continue to exert itself on the stress-linked clinical symptoms relationship at 6 months following baseline.

Aim 4: Moderating effects of resiliency in combination with a single hormone diathesis to predict stress-linked psychopathology at three months and six months from baseline.

4A. Investigate the moderation of baseline resiliency and cortisol on the positive association between high stress and risk of psychopathology at 3-month and 6-month follow-up.

4B. Investigate the moderation of baseline resiliency and testosterone levels on the positive association between high stress and risk of psychopathology at 3-month and 6-month follow-up.

Hypotheses for Aim 4

4A. Similar to the effects of social support and cortisol, high levels of resiliency will blunt the pathogenic effect of high cortisol on stress-linked clinical symptoms at three months following baseline. Specifically, among first responders with high cortisol levels, those with high resiliency will have lower symptoms than their low resiliency social support peers due to the stress-buffering of resiliency. It is expected that this joint moderation will persist at six months following baseline.

4B. Similar to the effects of social support and testosterone, high levels of resiliency will interact with high levels of testosterone to predict decreased symptoms at three months following baseline. Specifically, among first responders with high testosterone levels, those with high resiliency levels will have fewer symptoms than those their low resiliency peers due to stress-buffering of resiliency. This effect will continue to exert itself on the stress-linked clinical symptoms at 6 months following baseline.

Chapter 3: Methodology

Participant Recruitment

All participants were recruited from a local emergency medical service (EMS) within a large Southwestern city in the United States. Recruitment was conducted following seven continuing education (CE) meetings required for staff training in November and December 2013. Each first responder attended only one CE meeting. Inclusion criteria for the field study were employment by the local city-county EMS and attendance at staff CE training.

Researchers explained to potential participants the goals of the study and reinforced that participation in the study was voluntary and confidential. To reduce potential threat of coercive participation resulting from perceived pressure to participate from supervisors, EMS leadership personnel in each session reiterated the that study participation was optional and then left the training room. During an oral presentation by lead investigators, prospective participants were invited to participate in a study examining factors associated with stressors, health behaviors, and mental health in first responders. Researchers assured first responders that declining to participate or withdrawing from participation at any time would have no negative consequences. Furthermore, researchers stated that participation and data collected from this study would not be connected to or recorded in the participant's EMS employment record. Data for each participant would be tracked longitudinally to the 3-month follow-up and 6-month follow-up using a specifically assigned data identification number. Researchers explained that the documentation linking identification numbers to participant names would be kept securely locked and apart from all other securely locked data and securely destroyed at the completion of data collection. All study methodology –

participant recruitment and data collection – was approved by University of Texas Institutional Review Board (IRB #2013-03-0059).

DATA COLLECTION

Baseline data collection occurred at each of the seven CE sessions (collectively considered together as baseline data collection) across a period of three weeks, where participants completed a battery of self-report measures following full informed consent. After completion of the measures, participants provided two saliva samples to be assayed for cortisol and testosterone concentrations.

Clinical assessments of mental health symptom inventories were conducted repeatedly at three months and six months following baseline assessment. Clinical symptom data were collected via Qualtrics, a secure online survey platform approved by the University of Texas at Austin Institutional Review Board, to each participant's preferred email address given voluntarily by participants at baseline data collection for follow-up assessments. No email address was connected with participant data, rather electronic survey data were only linked with participant study identification number.

Hormone Data Collection

During baseline data collection, participants provided two saliva samples for hormonal analysis. Saliva collection procedures for hormone analysis followed previously established procedures from Josephs et al., 2012. Participants were given sealed saliva collection tubes within a plastic bag. The researcher asked the participant to unscrew the cap on each small collection tube and to place the end of a sterile straw in the participant's mouth while placing the other end of the straw into the collection tube. Then, the researcher

instructed the participant to imagine their favorite food while the participant tilted their head forward and allowed saliva to flow down the straw into the collection tube. Participants continued this process until saliva reached the 2mL line on the collection tube and secured back the collection tube cap. This same procedure was completed for both collection tubes. The two saliva samples were collected 20-30 minutes apart from one another to account for endocrine variability throughout the sessions. The two collection tubes were sealed within individual participant plastic bags, transported back to the University of Texas at Austin Psychology Department and immediately frozen and stored at -80^{o}C until analysis.

Psychological Data Collection

Participants completed the following measures during baseline data collection along with basic demographics (Appendix A).

Perceived Stress

An assessment of subjective stress was collected during each time point. Perceived stress was measured using the 10-item Perceived Social Stress Scale (PSS; Cohen, et al., 1983; α = .84 - .86). Participants responded to statements on thoughts and feelings related to general life stress in the past month (e.g., *In the last month, how often have you felt that you were on top of things?*) by indicating on a Likert-type response scale if they have experienced the statement from "never" to "very often." (Appendix B).

Critical Incidents

Another stress variable collected during the study was an objective measure of stress from EMS leadership represented by total number of ambulance calls responded to by each first responder. Calls were recorded by severity classified as Alpha, Bravo, Charlie,

Delta, and Echo with Echo rated calls being most severe. Calls were tallied monthly per responder by EMS leadership. These data were provided to study researchers to link with self-reported stress and mental health data for study participants by linking with the responder's badge number. Call totals were grouped into 3-month periods. For baseline collection data, calls were summed for the three months prior to the baseline data collection (across the seven CE sessions). For 3-month follow-up, calls were summed for the three months between baseline data collection and 3-month survey follow-up. For 6-month follow-up, calls were totaled for the 3 months between the 3-month survey delivery and 6-month survey delivery.

Social Support

Perceived social support was measured at baseline using the Interpersonal Support Evaluation (ISEL; Cohen & Hoberman, 1983; α = .77). Participants responded to 12 statements rating perceived quality of social relationships and supportive resources (e.g., *If I was stranded 10 miles from home, there is someone I could call who could come and get me.*) by indicating on a Likert-type response scale if examples of perceived support are definitely true to definitely false (Appendix C).

Resiliency

Resiliency was assessed during baseline data collection using the Brief Resiliency Scale (BRS; Smith et al., 2008; α = .80 - .91). Participants responded to 6 statements rating their perceived resiliency or hardiness (e.g., *I tend to bounce back quickly after hard times.*) by indicating on a Likert-type agreement scale (Appendix D).

Participants completed the following measures at baseline data collection and during electronic survey data collection at 3 months and 6 months following baseline.

Clinical Symptom Inventories

Clinical symptom inventories were assessed at each time point of data collection. PTSD symptoms were measured by the 17-item Post-Traumatic Stress Disorder Checklist – Civilian Version (PCL-C; Conybeare, Behar, Solomon, Newman, & Borkovec, 2012; α = .92 - .94). Participants evaluated severity of PTSD symptoms (e.g., *feeling jumpy or easily startled*) on a Likert scale ranging from not at all to extremely over the last month (Appendix E). Depression symptoms were measured by the 10-item Center for Epidemiological Studies Depression Scale (CES-D; Kohout, Berkman, Evans, & Cornoni-Huntley, 1993; Andressen, Malmgren, Carter, & Patrick, 1994; α = .73 - .86). Participants evaluated frequency of depressive symptoms (e.g., *I could not get* going) on a Likert scale ranging from rarely/never to most or all of the time on how they felt over the past week (Appendix F). Anxiety symptoms were measured using the 21-item Beck Anxiety Inventory (BAI; Beck & Steer, 1991; α = .92 from Beck, Epstein, Brown, & Steer, 1988). Participants evaluated severity of symptoms of anxiety (e.g., *heart pounding or racing)* on a Likert scale ranging from not at all to severely over the last week.

Clinical Health Behavior Inventories

Health behaviors were assessed at each time point of data collection. Alcohol use was assessed using a modified version of the Daily Drinking Questionnaire (DDQ; Collins, Parks & Marlatt, 1985; α = .73 from Lewis & Neighbors, 2004) (Appendix G). Sleep quality was assessed using the Pittsburgh Sleep Quality Inventory (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989; α = .91). Participants indicated subjective

quality and quantity of sleep problems over the past month (e.g., *how would you rate your sleep quality overall* or *how often have you had trouble sleeping because you feel too hot*) on a Likert scale (Appendix H).

Chapter 4: Statistical Analyses

HORMONE DATA ANALYSES

Hormone Data Selection

Salivary samples collected at baseline were analyzed for cortisol and testosterone concentrations. Upon arrival, all saliva samples were immediately frozen following collection and stored at -80°C until analysis. Salivary cortisol and testosterone concentrations were analyzed in-house with commercially available DRG, International enzyme immunoassay kits (DRG International, Springfield, NJ). Saliva samples were thawed completely and centrifuged for 15 minutes at 3000 rpm immediately prior to assay. The first and second saliva samples were assayed in duplicate for cortisol and testosterone, and hormone concentrations were averaged across the duplicate concentration values if the intra-assay CV was within the acceptable range (below 15%). If any sample generated a CV above 15%, the sample was re-assayed. Total intra-assay CV for cortisol across the entire sample was 4.04%, and total intra-assay CV for testosterone across the entire sample was 8.24%. To calculate a basal cortisol and testosterone concentration for each responder, the mean cortisol concentrations and testosterone concentrations were taken across both saliva samples.

Hormone Data Transformations

Significant variability is seen in the diurnal hormone concentrations of cortisol (Hansen, Garde, & Persson, 2008). Endogenous cortisol has a diurnal pattern in humans based on circadian rhythms. Cortisol peaks at its highest level approximately 30-minutes to 1-hour following awakening, known as the cortisol awakening response (CAR), and gradually descends throughout the day (Liening, Stanton, Saini, & Schultheiss, 2010;

Almeida, Piazza, & Stawksi, 2009). Given that hormone collection was conducted at different times of day depending on the scheduled time of the CE session (to accommodate the responders' different shift work schedules), statistical checks for differences between time of day of collection were calculated. There was a significant main effect for time of day of collection for cortisol, $F(1, 187) = 11.69$, $p = < 0.001$, where with highest concentrations occurring in the morning CE sessions, followed by the mean concentrations from afternoon CE sessions, and lastly the evening CE session concentrations (Figure 4). Gender differences in basal cortisol were assessed as well, but were not significant, $F(1, 188) = 0.117$, $p = 0.733$ (Figure 5), as has been shown in prior research (Kirschbaum, Wust, & Hellhammer, 1992). No gender by time of day collections interaction effects were found, $F(2, 184) = 0.152$, $p = 0.859$. To account for the main effect of collection time of day, z-score transformations of basal cortisol concentrations (ng/mL) were conducted within collection time of day subgroups.

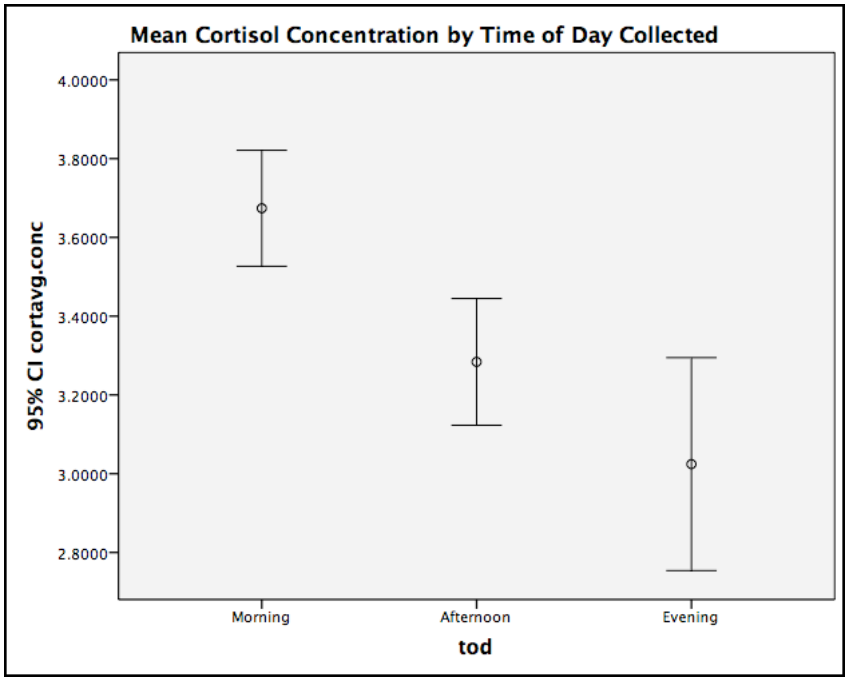

Figure 4. Mean Basal Cortisol Concentrations by Time of Day Collection.

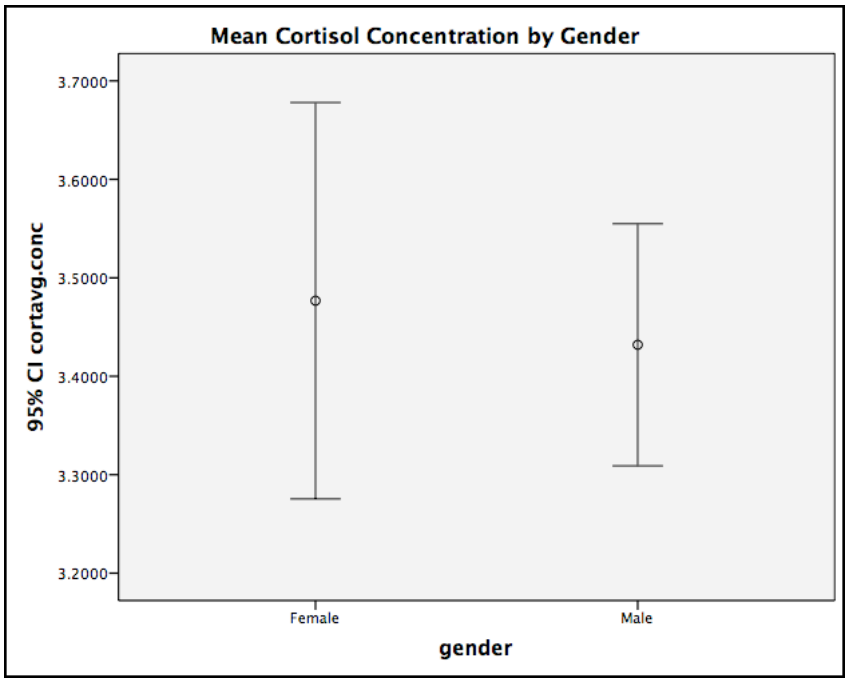

Figure 5. Mean Basal Cortisol Concentrations by Gender.

Sexually dimorphic concentrations of testosterone (Granger, Shirtcliff, Zahn-Waxler, Usher, & Klimes-Dougan, 2003) were expected to be observed within the study sample. There was a significant main effect for gender for basal testosterone, $F(1, 188) = 88.93$, $p = <0.001$, where men's testosterone concentrations were significantly higher than women's levels as predicted (Figure 6). There was no main effect of hormone collection time of day, $F(2, 187) = 1.706$, $p = 0.184$, as testosterone means did not significantly vary across morning, afternoon, and evening CE sessions (Figure 7). There was no significant interaction between gender and time of day collection for testosterone levels, $F(2, 184) = 0.21$, $p = 0.811$. To account for the main effect of gender within raw testosterone concentrations (pg/mL), z-score transformations of basal testosterone were conducted within gender groups.

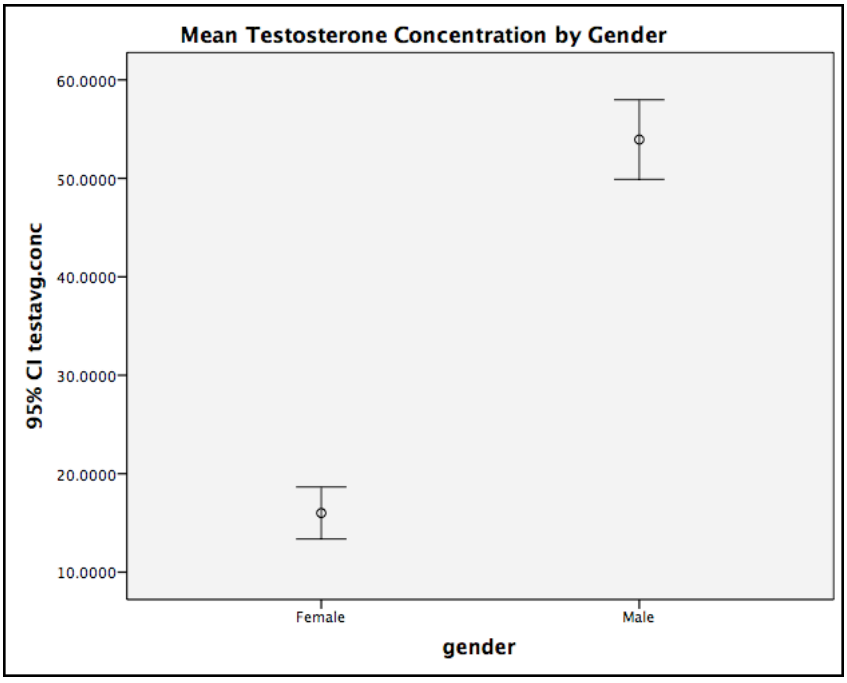

Figure 6. Mean Basal Testosterone Concentrations by Gender.

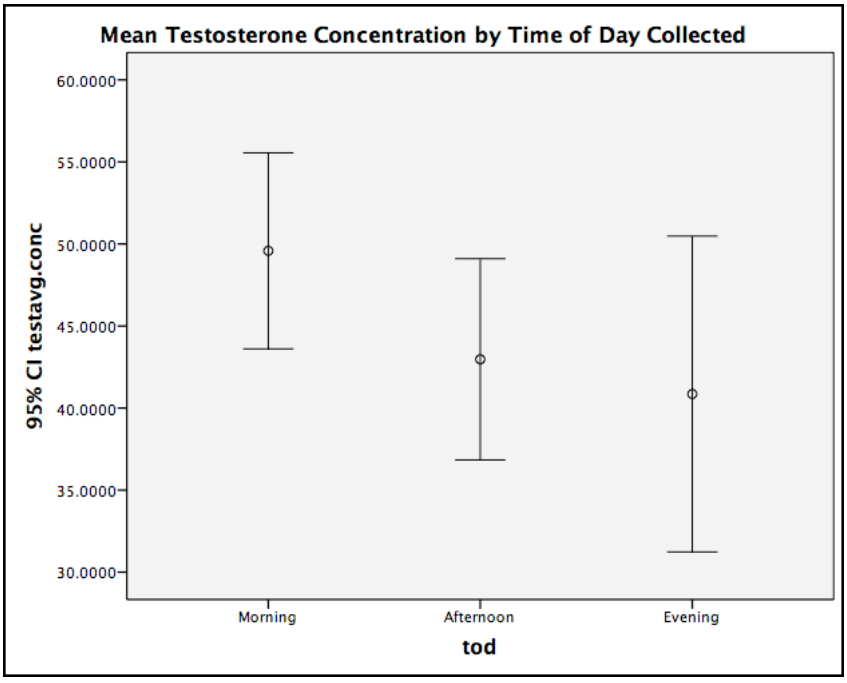

Figure 7. Mean Basal Testosterone Concentrations by Time of Day Collected.

PREDICTION MODELS

Ordinary least squares multivariate linear regression was the statistical technique chosen to estimate these regression parameters. Another regression modeling technique known as mixed effect modeling or multilevel regression (MLR; Hox & Stoel, 2005) would also be appropriate to model these predictors across the 6-month study time frame. An advantage to MLR is that it can include more than one error variable in the regression as random effects, while fixed effects remain very similarly calculated as is done in OLS. MLR also allows for hierarchal or nested data modeling. For example, within this sample, MLR could predict effects within the individual first responder at one level as well as effects for the entire group of local first responders. The random effect terms in MLR are capable of predicting outcome variables while accounting for multiple repeated measures within a sample. Despite these advantages of MLR, OLS regression modeling was chosen as the regression methodology for this dissertation as OLS model estimation was still adequately able to predict clinical outcome scores based on baseline hormone and stress predictors variables at 3-month and 6-month follow-up.

Fixed Effect Predictors

The fixed effects included within the OLS models (i.e., the predictor variables) predicting psychopathology included baseline cortisol, baseline testosterone, baseline perceived social support, baseline resiliency, and baseline perceived stress. Additionally, baseline psychopathology scores (.e.g. PTSD symptoms) were added to the model to account for the starting severity of each responder on the clinical outcome. All models were

built following the diathesis-stress theoretical framework with a priori hypotheses. First, single hormone models were built with perceived stress effects. Then a dual hormone – cortisol and testosterone – model on perceived stress was built. The remaining four models added one stress-buffering factor (either social support or resiliency) into the single hormone and perceived stress models. Of note, when social support or resiliency was modeled into the hormone-moderated stress models, only one hormone (cortisol *or* testosterone) was included as a predictor to limit the interpretation of potential interactive effects to three predictor variables.

Seventy linear regression models were conducted. For each of the five outcomes, seven models were conducted to predict psychopathology at 3-month follow-up. The same model predictors were then used to predict the same clinical outcome at 6-month follow-up. All statistical modeling was conducted in R (R Core Team, 2013). To account for the high number of regression models conducted, which inflates the Type I error probability of falsely rejecting the null hypothesis, a multiple comparison technique of a Bonferroni-corrected adjusted *p* value, was used to evaluate statistical significance. This was calculated by dividing the Type I error probability (0.05) by the number of models (70) which produced an adjusted *p* value of 0.000714.

Multivariate regression assumptions of normality, independence, and constancy of variance were tested for each model. Regression assumptions of residual error independence and constancy of variance (i.e., homoscedasticity) were met. Independence of residuals was analyzed using the Durbin-Watson tests for autocorrelations of the residuals which revealed none. Constancy of variance was analyzed by visually graphing scatterplots comparing model residuals with model fitted values to ensure residual values are generally equally scattered across for all predicted (fitted) values. However, all models except the sleep disturbance models did not meet the criterion of normally distributed

residuals tested by the Shapiro-Wilkes Test of Normality. To attempt correction of this regression violation, any model that closely met ($p < 0.001$) the adjusted Bonferroni p value was then tested for outliers. One model met this criterion of approximate Type 1 error probability close to the Bonferroni-correct p value. Two outlier cases were removed, and the model was conducted again with outlier cases excluded; while normality of the residuals improved with exclusion of the outliers, it still did not pass the Shapiro-Wilkes Test of Normality nor meet Bonferroni-corrected statistical significance to be an interpretable effect.

Any hypothesized second-degree and third-degree interactions of predictors found to have statistical significance within the prediction models were planned to be further analyzed post-hoc. In order to better visualize significant interactions, simple slopes analyses based on Aiken, West, and Reno's (1991) statistical procedure was used to center variables at -1 and +1 standard deviations from their respective means in order to constrain values of continuous variables into discrete levels to better visualize the moderating interaction. This methodology's constraint of a continuous variable into a discrete variable by using arbitrary cut points is recognized as restrictive toward identifying interaction effects within the literature (Harrell Jr. & Slaughter, 2017).

The use of the Johnson-Neyman (JN) technique of identifying regions of significance has been around since the mid-1930's but has been used over the years to more fully interpret moderating effects of continuous variables (Johnson & Neyman, 1936; Rast, Rush, Piccinin, & Hofer, 2014). The J-N technique identifies conditions under which moderators have a significant effect on an outcome variable and the conditions where these effect are not significant. In other words, this technique computes the exact values for which a conditional effect of a moderator is significant and where it is not. This technique avoids losing detection of any significant conditional effects of a moderator that may not

be captured by the arbitrary categorization of predictor variables at -1/+1 standard deviations or other predetermined cut points such is done by Aiken, West, & Reno (1991) simple slope analyses.

Chapter 5: Results

SAMPLE DEMOGRAPHICS

Baseline data collection, which included both endocrine *or* survey data, was completed at baseline for 204 participants. Fourteen participants completed only hormone samples and did not complete the any survey data. As data models included both hormone and survey data as predictors, these 14 participants were removed from the dataset bringing the *N* of the study to 190 for baseline data collection. All further analyses were conducted using *N*=190 dataset. The baseline sample was 77.9% male. The mean of the baseline sample was 35.3 years (*SD* = 8.2 years). Reported racial identities of the baseline sample were 89.7% White, 1.5% Black or African-American, 1.5% Asian or Asian-American, 0.5% Native or Pacific Islander, 3% multiracial, 3.4% other racial identity, and 0.5% chose not to answer. Distribution of highest level of education achieved was 41.7% some college undergraduate coursework (no degree), 27.9% associate degree, 25.0% bachelor's degree, 3.4% some graduate coursework (no degree), 1.5% graduate degree, and 0.5% chose not to answer. The sample consisted of 55.9% paramedics, 16.7% EMTs, 14.2% captains, 7.4% commanders, 4.4% 911 call operators, and the remaining 1.5% serving in dual roles within the above positions. One quarter (25.5%) of participants self-reported an annual salary range between $45,000 to $59,999 while another quarter (24.0%) of the sample reported an annual salary between $60,000 to $74,999; annual salaries reported ranged between less than $15,000 to greater than $90,000.

A bimodal distribution of time served employed with the agency was noted. The modal range of years of service was 11-15 years (26.0%), followed closely by 6-10 years (25.5%). The next highest frequencies for years of service were 1-3 years (17.2%) and less than one year on the job (15.7%), suggesting a frequent drop-off in employment following

3 years on the job as well as greater than 15 years on the job. The overwhelming majority (89.7%) of our sample at baseline reported not currently receiving any treatment or general support from a mental health professional.

To ease time demands on the participants at follow-up timepoints, no repeat assessments of demographics were collected. Therefore, we were unable to assess for changes in other reported demographics (annual salary, current position, utilization of mental health resources) across the 6-month time frame. Nevertheless, hypotheses did not rely on demographically-stratified subgroups within the sample, but rather explores within the sample as a whole. Limitations of this approach and ideas for further work that emphasize inclusion of richer demographic data in modeling will be discussed in the study discussion.

Sample Attrition

At the 3-month data collection timepoint, 158 participants completed the online survey data collection – a 16.8% attrition of study participants from the baseline data collection. At the 6-month data collection timepoint, 111 participants completed the online survey data collection – a 41.5% attrition rate from baseline data collection. Only these participants were used to compare 3-month (N=158) and 6-month (N=111) mental health outcome scores with baseline data, respectively; therefore, any participant with missing outcome data at the timepoint being predicted (i.e., at 3-month or 6-month follow-up) was excluded from model estimation.

Non-Responder Analyses

Non-responder analyses were completed within baseline data to ensure no significant differences existed in the predictor and outcome variables between those

participants who completed either follow-up data collection (i.e., 3-month or 6-month follow-up) and those who chose to not provide follow-up survey data. Independent Student's t-tests comparing the two groups (follow-up responders vs. non-responders) across the four predictor variables (cortisol, testosterone, social support, and resiliency) were conducted. No significant differences were found between follow-up study participants and non-responders in cortisol concentration (t = 1.272, df = 194, p = 0.205), testosterone concentration (t = 0.147, df = 194, p = 0.883), social support (t = -1.237, df = 186, p = 0.218), and resiliency (t = 0.059, df = 187, p = 0.953). The perceived stress variable did not significantly differ between those who participated in follow-up assessments compared to those who did not (t = 0.706, df = 187, p = 0.481). These null findings argue that no individual diatheses and perceived stress within the sample could predict who would respond at later data collections versus who would not respond, strengthening the generalizability of our prediction models to the first responder population overall.

Finally, all baseline outcome variables were analyzed to ensure no significant differences existed in baseline data for responders and non-responders to longitudinal data collection. All analyses were nonsignificant – PTSD (t = 1.661, df = 187, p = 0.098), depression (t = 1.449, df = 166, p = 0.149), anxiety (t = 1.047, df = 188, p = 0.296), alcohol use (t = 0.800, df = 185, p = 0.425), and sleep disturbance (t = 1.085, df = 144, p = 0.280). Generalizability of these prediction models is strengthened as those first responders included in our model predictions did not significant differ in our four predictor diatheses, perceived stress variable, and five mental health symptoms and behaviors than those excluded from regression model predictions.

SAMPLE DESCRIPTIVE STATISTICS

Table 1 shows the means, standard deviations, and sample size of each of the predictor variables (top-half of the table) at baseline, and the means, standard deviations, and sample size of each outcome variable (bottom-half of the table) at baseline, 3-month follow-up and 6-month follow-up. Mean outcome scores did change but not significantly across data collection for the sample as a whole. However, the number of respondents per follow-up time period consistently dropped throughout the study.

	Baseline			**3-Month Follow-Up**			**6-Month Follow-Up**		
Variable	*M*	*SD*	*N*	*M*	*SD*	*N*	*M*	*SD*	*N*
Cortisol (ng/mL)	3.44	0.73	190	-	-	-	-	-	-
Testosterone (pg/mL)	45.95	27.36	190	-	-	-	-	-	-
Perceived Stress	15.01	6.66	189	-	-	-	-	-	-
Social Support	40.45	6.46	188	-	-	-	-	-	-
Resiliency	4.01	0.75	189	-	-	-	-	-	-
PTSD	28.93	11.45	189	28.85	13.33	126	29.30	14.22	83
Depression	7.67	5.30	168	9.67	5.79	126	9.79	6.52	91
Anxiety	6.77	6.55	190	7.76	7.99	128	8.55	9.90	93
Weekly Alcohol Use	8.37	10.98	188	15.70	22.12	114	14.80	13.54	84
Sleep Quality	7.64	3.726	146	7.92	4.301	119	7.98	4.31	90

Table 1. Descriptive Statistics of Predictor and Outcome Variables.

Bivariate correlations of each baseline predictor variable were conducted (Table 2). Cortisol and testosterone were positively associated with each other (p = 0.001). Testosterone and social support were negatively associated (p = 0.011). Perceived stress was negatively associated with social support (p = < 0.001) and resiliency (p = < 0.001) as theoretically supported. Social support and resiliency were positively correlated (p = <0.001).

Predictor	Cortisol	Testosterone	Perceived	Social	Resiliency

Variables:			Stress	Support	
Cortisol	1.00	--	--	--	--
Testosterone	.239**	1.00	--	--	--
Perceived Stress	.060	.093	1.00	--	--
Social Support	-.099	-.184*	-.474***	1.00	--
Resiliency	-.009	-.135	-.611***	0.465**	1.00

Note: * $p < .05$; ** $p < .01$; *** $p < .001$

Table 2. Correlations of Predictor Variables at Baseline.

Bivariate Correlations were also conducted for each outcome variable across all data collection time points – i.e., baseline, 3-month follow-up, and 6-month follow-up (Table 3). All outcome variables were strongly positively associated with the other two collection timepoint scores (all *p*'s = <0.01). For instance, depression scores at 6 months were positively correlated to baseline depression and 3-month depression scores. Given this strong correlation, baseline values of all outcomes were added as a control to all regression models to help account for some shared variance.

	Baseline	3 Months	6 Months
PTSD			
Baseline	1.0	--	--
3 Months	0.643***	1.0	--
6 Months	0.631***	.877***	1.0
Depression			
Baseline	1.0	--	--
3 Months	0.714***	1.0	--
6 Months	0.629***	.826***	1.0
Anxiety			
Baseline	1.0	--	--
3 Months	0.655***	1.0	--
6 Months	0.667***	.802***	1.0
Weekly Alcohol Use			
Baseline	1.0	--	--
3 Months	0.313**	1.0	--
6 Months	0.663***	.687***	1.0

Sleep Quality			
Baseline	1.0	--	--
3 Months	0.726***	1.0	--
6 Months	0.698***	.798***	1.0

Note: ** p < .01, *** p < .001

Table 3. Correlations of Outcomes Variables across Data Collection Timepoints.

DIATHESES-STRESS REGRESSION MODELS

To simplify data reporting of all seventy prediction models results were grouped by combination of diatheses (e.g. cortisol-only models or cortisol and social support) predicting stress-linked psychopathology. For each subset of like-diathesis models, all five clinical symptoms and health behaviors models are shown: PTSD, depression, anxiety, substance use, and sleep quality. For each outcome variable, a model for each combination of diatheses is shown to predict symptoms three months later, as well as, a second model to predict symptoms six months since baseline data were collected. Perceived stress was measured during baseline data collection. Full regression data tables are included as supplementary to this dissertation (Appendix I). Statistical significance was considered only if p value met the adjusted Bonferroni correction for multiple comparisons ($p < 0.000714$). As seventy models were run, using a standard 0.05 type I error probability would have predicted significance be found in 5% (approximately 3-4 models total) based on random error variance alone. Given this stringent adjusted p-value used to avoid Type 1 error null hypothesis rejections, no significant effects were found across the study as no hypothesized effects reached statistical significance at the adjusted p value. No interactions were further analyzed post-hoc using simple slopes analysis or JN regions of significance as no Bonferroni adjusted statistical significance was met.

Cortisol-Only Models (Study Aim 1A)

Hypothesis 1A: High baseline cortisol will interact with high levels of stress to predict increased clinical symptoms at three months following baseline. It is expected that this interaction of cortisol and high stress will continue to be impactful at six months following baseline.

Contrary to hypotheses, at 3-month follow-up basal cortisol did not interact with baseline perceived stress to predict clinical symptoms of PTSD (β = 0.11, p = 0.479); depression (β = 0.11, p = 0.084); anxiety (β = 0.12, p = 0.15); weekly alcohol use (β =0.19, p = 0.557); and sleep disturbance (β = 0.08, p = 0.133). Non-significance of cortisol's single-hormone moderation continued at 6-month follow-up for PTSD (β = 0.02, p =0.908); depression (β = -0.02, p = 0.778); anxiety (β =0.10, p = 0.379); weekly alcohol use (β = -0.09, p = 0.627); and sleep disturbance (β = 0.08, p = .247).

Testosterone-Only Models (Study Aim 1B)

Hypothesis 1B: High baseline testosterone will interact with high levels of stress to predict decreased clinical symptoms at three months following baseline, and its effects are expected to persist at six months following baseline.

Contrary to hypotheses, at 3-month follow-up basal testosterone did not interact with baseline perceived stress to predict clinical symptoms of PTSD (β = <0.01, p = 0.968); depression (β = -0.06, p =0.188); anxiety (β = -0.06, p = 0.330); weekly alcohol use (β = -0.24, p = 0.341); and sleep disturbance (β = <0.01, p = 0.952). The pattern of non-significance for basal testosterone's single-hormone moderation was observed at 6-month

follow-up for PTSD (β = -0.09, p = 0.608); depression (β = -0.04, p = 0.542); anxiety (β = -0.13, p = 0.124); weekly alcohol use (β = -0.31, p = 0.049); and sleep disturbance (β = -0.08, p = 0.195).

Dual Hormone (Cortisol and Testosterone) Models (Study Aim 2)

Hypothesis 2: High baseline testosterone will blunt the pathogenic effect of high cortisol on stress-linked clinical symptoms at three months following baseline. Specifically, among first responders with high cortisol levels, those with also high testosterone levels will report lower symptoms than their low-testosterone peers due to the stress-buffering effects of testosterone. It is expected that this joint moderation will persist at six months following baseline.

Contrary to hypotheses, at 3-month follow-up basal cortisol and testosterone did not interact with baseline perceived stress to predict clinical symptoms of PTSD (β = -0.14, p = 0.471); depression (β = -0.05, p =0.529); anxiety (β = 0.15, p = 0.170); weekly alcohol use (β = -0.07, p = 0.874); and sleep disturbance (β = 0.02, p = 0.783). The pattern of non-significance for cortisol and testosterone's dual moderation was observed at 6-month follow-up for PTSD (β = 0.02, p = 0.910); depression (β = 0.07, p = 0.427); anxiety (β = 0.03, p = 0.837); weekly alcohol use (β = -0.11, p = 0.560); and sleep disturbance (β = -0.01, p = 0.934).

Cortisol and Social Support Models (Study Aim 3A)

Hypothesis 3A: High levels of social support will blunt the pathogenic effect of high cortisol on stress-linked clinical symptoms at three months following baseline. Specifically, among first responders with high cortisol levels, those with high social support levels will have lower symptoms than their low social support peers due to the stress-buffering of social support. It is expected that this joint moderation will persist at six months following baseline

Contrary to hypotheses, at 3-month follow-up basal cortisol and baseline social support levels did not significantly moderate baseline perceived stress on clinical symptoms of PTSD (β = -0.02, p = 0.451); depression (β = 0.01, p = 0.105); anxiety (β = -0.04, p = 0.000783); weekly alcohol use (β = 0.06, p = 0.229); and sleep disturbance (β = <0.01, p = 0.548). Given the close significance of the 3-month anxiety symptoms model and non-normality of residuals, outliers were tested, and two cases were removed from the model to improve the normality assumption of the residuals for linear regression. The regression coefficient of the interaction of this outlier-excluded model is reported above. This model with outlier cases excluded still did not reach adjusted p-value significance criterion (p = 0.000714). Similarly, at 6-month follow-up baseline cortisol and social support moderation of perceived stress was not observed in the separate prediction models for PTSD (β = -0.03, p = 0.296); depression (β = 0.02, p = 0.131); anxiety (β = -0.03, p = 0.090); weekly alcohol use (β = 0.04, p = .105); and sleep disturbance (β = <0.01, p = 0.738).

Testosterone and Social Support Models (Study Aim 3B)

Hypothesis 3B: High levels of social support will interact with high levels of testosterone to predict decreased symptoms at three months following baseline. Specifically, among first responders with high testosterone levels, those with high social support levels will predict even fewer symptoms than those their low social support peers due to social support's stress buffering. This effect will continue to exert itself on the stress-linked clinical symptoms relationship at 6 months following baseline.

Contrary to hypotheses, at 3-month follow-up basal testosterone and baseline social support did not significantly moderate baseline perceived stress on clinical symptoms of PTSD (β = -0.02, p = 0.306); depression (β = 0.01, p = 0.187); anxiety (β = -0.02, p = 0.071); weekly alcohol use (β = -0.02, p = 0.657); and sleep disturbance (β = <0.01, p = 0.753). Similarly, at 6-month follow-up no basal testosterone and social support moderation of baseline perceived stress was observed in the separate prediction models for PTSD (β = -0.032 p = 0.547); depression (β = 0.01, p = 0.613); anxiety (β = 0.01, p = 0.502); weekly alcohol use (β = -0.01, p = 0.672); and sleep disturbance (β = -0.02, p = 0.163).

Cortisol and Resiliency Models (Study Aim 4A)

Hypothesis 4A: Similar to the effects of social support and cortisol, high levels of resiliency will blunt the pathogenic effect of high cortisol on stress-linked clinical symptoms at three months following baseline. Specifically, among first responders with high cortisol levels, those with high resiliency will have lower symptoms than their low

resiliency social support peers due to the stress-buffering of resiliency. It is expected that this joint moderation will persist at six months following baseline.

Contrary to hypotheses, at 3-month follow-up basal cortisol and baseline resiliency did not significantly moderate baseline perceived stress on clinical symptoms for PTSD (β = -0.17, p = 0.379); depression (β = 0.08, p = 0.312); anxiety (β = -0.12, p = 0.250); weekly alcohol use (β = 0.02, p = 0.970); and sleep disturbance (β = -0.03, p = 0.688). Similarly, at 6-month follow-up baseline cortisol and resiliency moderation of perceived stress was not observed in the separate prediction models for PTSD (β = -0.14, p = 0.553); depression (β = 0.05, p = 0.644); anxiety (β = -0.21, p = 0.139); weekly alcohol use (β = 0.13, p = .573); and sleep disturbance (β = -0.06, p = 0.548).

Testosterone and Resiliency Models (Study Aim 4B)

Hypothesis 4B: Similar to the effects of social support and testosterone, high levels of resiliency will interact with high levels of testosterone to predict decreased symptoms at three months following baseline. Specifically, among first responders with high testosterone levels, those with high resiliency levels will have fewer symptoms than those their low resiliency peers due to stress-buffering of resiliency. This effect will continue to exert itself on the stress-linked clinical symptoms at 6 months following baseline.

No hypothesized findings were supported for the final study aim regarding testosterone and resiliency. No baseline perceived stress moderation by an interaction of testosterone and resiliency ratings were found on clinical symptom reporting at 3-month follow-up for

PTSD (β = -0.12, p = 0.425); depression (β = 0.14, p = 0.017); anxiety (β = -0.08, p = 0.335); weekly alcohol use (β = -0.01, p = 0.977); and sleep disturbance (β = -0.01, p = 0.919). Similarly, at 6-month follow-up, no significant moderation of baseline perceived stress by testosterone and resiliency was found across the clinical prediction model for PTSD (β = 0.11, p = 0.586); depression (β = 0.11, p = 0.210); anxiety (β = 0.10, p = 0.366); weekly alcohol use (β = -0.16, p = 0.417); and sleep disturbance (β = 0.03, p = 0.739).

SUPPLEMENTARY RESULTS: CRITICAL INCIDENTS AS PREDICTORS

Given that critical incident data was collected for each participant during the three months prior to baseline data collection, exploratory analyses were conducted to see if total number of critical incidents (represented by the number of field calls worked) or separate totals of a severity category for critical incident calls was predictive of the baseline variables. OLS regressions for each classification of incident call (Alpha through Echo severity) and total number of incident calls overall were separately conducted to see if they predicted baseline cortisol, baseline testosterone, baseline social support, baseline resiliency, and baseline perceived stress. In addition, OLS regressions were conducted to see if the number of incident calls in the three months prior to baseline data collection was associated with baseline clinical outcomes for PTSD, depression, anxiety, alcohol use, and sleep quality.

For predicting baseline cortisol concentration, a main effect for Alpha calls was found (β = -0.030, p = 0.032, r = 0.15) where the lower number of Alpha calls responded to predicted lowered basal cortisol levels. No main effects were found for Bravo calls (β = -0.001, p = 0.757), Charlie calls (β = 0.019, p = 0.104), Delta calls (β = 0.007, p = 0.390), Echo calls (β = -0.003, p = 0.759), nor Total calls (β = <0.001, p = 0.788) in the

three months prior to baseline. For predicting baseline testosterone levels, no main effects were found for Alpha calls (β = -0.014, p = 0.320), Bravo calls (β = -0.003, p = 0.594), Charlie calls (β = 0.008, p = 0.487), Delta calls (β = 0.004, p = 0.585), Echo calls (β = < 0.001, p = 0.968), nor Total calls (β = <0.001, p = 0.589) in the three months prior to baseline.

For predicting baseline social support levels, a main effect for Total Calls (β = 0.011, p = 0.031, r = 0.15) and a trending main effect for Echo calls was found (β = 0.121, p = 0.073, r = 0.13), where higher number of Echo or Total calls worked by the participant, the higher the reported social support level at baseline. No main effects for baseline social support were found for Alpha calls (β = -0.142, p = 0.126), Bravo calls (β = 0.057, p = 0.135), Charlie calls (β = 0.078, p = 0.315), nor Delta calls (β = -0.045, p = 0.412). For predicting baseline resiliency in the responders, a main effect for Alpha calls (β = -0.026, p = 0.015, r = 0.17) and Bravo calls (β = 0.009, p = 0.036, r = 0.15). Interestingly, increases in Alpha calls were associated with decreases in resiliency; however, increases in Bravo calls were also associated with increases in resiliency. It is not clear what mechanism would conceptualize this reversal of effects. No main effects were observed for Charlie calls (β = -0.005, p = 0.546), Delta calls (β = 0.003, p = 0.537), Echo calls (β = -0.001, p = 0.880), nor Total calls (β = <0.001, p = 0.125) on baseline resiliency.

For predicting baseline perceived stress levels, main effects existed for Alpha calls (β = 0.255, p = 0.007, r = 0.19) and Total calls (β = -0.011, p = 0.046, r = 0.14). The dual effects exert there influence on perceived stress ratings in opposite directions. While increases in Alpha calls are associated with increased baseline stress, decreases in Total calls are associated with lower baseline stress.

It was also worthwhile to see if a first responders exposure to incident calls (Total or by severity category) in the three months prior to baseline could affect his or her clinical

outcome score. For PTSD symptom predictions, there was only a main effect of Alpha calls (β = 0.454, p = 0.006, r = 0.19) where increases in number of Alpha calls was associated with increased PTSD symptoms reported at baseline. On the other hand, no main effects were found for Bravo calls (β = -0.091, p = 0.179), Charlie calls (β = -0.025, p = 0.851), Delta calls (β = -0.051, p = 0.597), Echo calls (β = -0.089, p = 0.458), nor Total calls (β = -0.007, p = 0.469).

To predict depression symptoms at baseline, only related predictors were Alpha calls (β = 0.265, p = <0.001, r = 0.25) and Bravo calls (β = -0.06, p = 0.037, r = 0.16) experienced in the three months prior to baseline. As with PTSD, we see that these call main effects exert their influence in different directions on depression symptoms. Higher Alpha calls is related to higher depression symptoms, but higher Bravo calls are expected to predict lower depression symptoms at baseline. No main effects were found for Charlie calls (β = -0.035, p = 0.595), Delta calls (β = 0.003, p = 0.936), Echo calls (β = -0.070, p = 0.217), nor Total calls (β = 0.004, p = 0.352).

To predict anxiety symptoms at baseline, only Alpha calls had a significant effect (β = 0.219, p = 0.020, r = 0.17) where a high number of Alpha calls worked in the three months prior to baseline, increased one's likelihood of reporting greater anxiety. No main effects were found for Bravo calls (β = -0.063, p = 0.106), Charlie calls (β = 0.071, p = 0.363), Delta calls (β = -0.035, p = 0.524), Echo calls (β = -0.065, p = 0.338), nor Total calls (β = -0.003, p = 0.480).

No main effects were found predicting weekly alcoholic drink consumption at baseline across all categorizations of calls: Alpha calls (β = 0.022, p = 0.889), Bravo calls (β = -0.085, p = 0.199), Charlie calls (β = 0.120, p = 0.368), Delta calls (β = 0.023, p = 0.805), Echo calls (β = 0.048, p = 0.680), and Total calls (β = -0.005, p = 0.564). Similar to alcohol use predictions, critical incidents calls in the three months prior to baseline had

no significant effect on sleep quality reported at baseline regardless of call category: Alpha calls ($\beta = 0.048$, $p = 0.451$), Bravo calls ($\beta = -0.023$, $p = 0.379$), Charlie calls ($\beta = -0.010$, $p = 0.839$), Delta calls ($\beta = 0.003$, $p = 0.928$), Echo calls ($\beta = 0.047$, $p = 0.274$), and Total calls ($\beta = -0.001$, $p = 0.652$).

Chapter 6: Discussion

SIGNIFICANT HORMONE FINDINGS

To test for stress moderation of five distinct diatheses (cortisol, testosterone, dual hormone effects (cortisol and testosterone), social support, and resiliency), baseline levels of perceived stress were tested for the presence of interactions with baseline levels of the five diatheses. No evidence of diathesis-stress moderation was found. Neither single nor dual hormone effects were found for PTSD, depression, anxiety, alcohol use, and sleep disturbance at either 3-month or 6-month follow-up (all *p's > 0.000714)*. This low adjusted *p* value which was derived from a Bonferroni correction for multiple comparisons of seventy regression models was used to reduce the probability of inflated Type 1 error.

No testosterone effects were found in this study, which stands in contrast to a large literature showing a significant association between high testosterone and improved mental health and well-being. Further, no cortisol effects were found, which is perhaps less surprising, given the mixed literature on cortisol and psychopathology.

Further, no dual hormone effects were found, which also stands in contrast to the admittedly-small literature on the joint effects of testosterone and cortisol on psychopathology (Glenn et al., 2011; Tackett et al., 2014; Josephs et al., 2017; Cobb et al, 2018). However, Josephs et al. (2017) only reported significant effects for hormone reactivity measures. It is possible that basal cortisol levels may not be appropriate for predicting PTSD symptoms but rather cortisol reactivity along with testosterone reactivity may be more appropriate in estimating the variance in symptom expression. (Klaassens, Giltay, Cuilpers, van Veen, & Zitman, 2012; Meewisse et al., 2007). In regard to depressive

symptoms, Cobb et al. (2018) found that the risky profile for downstream depression in soldiers with low testosterone and high testosterone reactivity to in theatre stressors (2018). Furthermore, high basal cortisol was protective against depression, which stands in opposition to this study's predicted hypotheses. Cobb et al.'s finding was also contrary to the cortisol literature linking hypercortisolism to increased depression (Burke et al., 2005; Herbert, 2013). However, there is evidence that low basal cortisol is predictive of earlier relapse in recurrent major depression (Bockting et al., 2012). However, in Cobb et al. (2018), this pattern reverses for cortisol reactivity effects on war-zone stress-evoked depression. High cortisol reactivity was associated with perceived stress to predict greater depression symptoms. It is possible that the current study's failure to find dual hormone effect can be partially attributed to not capturing both basal and reactivity concentrations of hormones to be modeled together as supported for PTSD and depression.

Tackett et al., (2014) reported that parent-reported externalizing behaviors, common diagnostic symptoms of many conduct and personality disorder especially of childhood and adolescence, were predicted by the three-way interaction between basal cortisol, basal testosterone levels, and the pathological personality traits of disagreeableness and emotional instability. Higher testosterone was associated with greater externalizing behaviors but only when cortisol was low, an opposite pattern of the hypothesized risky profile for the current study which expected that low testosterone and high basal cortisol would be associated with psychopathology. However, given that externalizing problems are approach behaviors and correlated with aggression, Tackett et al. (2014) did find the classic dual hormone pattern (Mehta & Prasad, 2015; Mehta & Josephs, 2010). Because, however, externalizing behaviors are social approach behaviors,

it follows that the internalizing symptoms and behavioral withdrawal prevalent in mood and anxiety disorders may be affected by the opposite endocrine profile (low testosterone with high cortisol), which was predicted by this study. Although I found no significant effects, the premise of reversing the externalizing symptoms dual hormone profile in further studies of psychopathology with significant internalizing symptoms remains a testable hypothesis.

LIMITATIONS

Arising from nonsignificant results across all hypotheses is the need to consider what occurred within the current study to prevent confirmation of any of the multiple dual hormone hypotheses. Although it is possible that these hypotheses were incorrect, there exists an established literature on the single-hormone effects of cortisol and testosterone in psychopathology and a growing literature of cortisol and testosterone's dual effects in clinical research and social behaviors. Other sample characteristics such as small sample size and significant study attrition (41.5%) by the end of the 6-month study timeframe likely contributed significantly to poor predictive power. Multiple regression models were constructed and analyzed to address each hypothesis for each clinical outcome in seventy regression models totals. This risk of Type 1 error inflation for false positive statistical significance of tested effect was corrected using Bonferroni's alpha correction ($p = 0.000714$). This adjusted significance only allowed for very small alpha (Type 1) probabilities and corresponding larger significant effect sizes (e.g., *r)* to report hypothesized effects. The magnitude of effect required to meet the Bonferroni criterion were not likely to be observed within this data sample. Additionally, the assumption of normality of the hypothesized models' residuals was not achieved in most models.

Graphical examination of the residuals' distribution showed non-normal distribution in the tails.

Outside of study characteristics, the hypothesized hormone-moderated mechanisms of cortisol and testosterone may not be generalizable to the current subset of psychopathology. Dual hormone moderation of testosterone and cortisol on stress may not be an overarching mechanism in maintaining any mood, anxiety, or trauma-related psychiatric disorder. Failure to find effects also may explain the mixed literature review found regarding cortisol and testosterone in the PTSD literature (Meewisse et al., 2007; Violanti et al., 2007; Mulchahey et al., 2001; Reijnen et al., 2015; Mason et al., 1990). In fact, Josephs et al. (2017) was the only study identified by this author to examine jointly testosterone and cortisol effects on PTSD. More research of the proposed mechanisms of these hormones within human and animal models of specific psychiatric disorders is needed rather than applying the same hormone risk profile of joint testosterone and cortisol as a general predictor of mood and anxiety psychopathology.

Additionally, alcohol use and sleep disturbance were both included in prediction models as outcome variables rather than predictor variables. Currently, sleep disruption and substance use are diagnostic symptoms in psychopathology of mood and anxiety disorders. However, given their behavioral nature, these factors are also very common intervention targets. Given that the inclusion of these factors as outcomes in our studies was not supported by the hypotheses, it is not expected that weekly alcohol consumption and sleep quality would be associated with basal cortisol and testosterone levels. Even after taking into account that strict adjusted *p* value and small sample size limited the probability of effect detection to only those with larger effect size, it is still unlikely that cortisol and testosterone would influence stress-linked sleep or alcohol use behaviors as separate main effects or a joint interaction. The silver lining of these non-significant findings is that they

provide evidence to continue to conceptualize alcohol or other substance use and sleep disturbance as behavioral predictors for psychopathology ratings. This is confirming of psychological interventions that focus on changing behaviors to result in changing symptom severity.

FUTURE STUDY DESIGNS

Future studies of dual hormone moderation of stress for psychopathology within the first responder population, and the general population as a whole, is warranted to first and foremost provide greater published findings to the mixed literature and to correct the dearth within the first responder field. However, measurement of basal cortisol and testosterone should be more tightly controlled for time of day of collection. Accounting for the individual's sleep schedule in the days before saliva collection and the individual's current shift work schedule is also needed. As cortisol diurnal patterns are impacted by sleep-wake schedules, controlling for sleep from the night before would be beneficial to eliminating variance within cortisol concentrations.

It would be interesting in future studies to explore how shift work within the first responder profession has impacted the cortisol awakening response (CAR) and diurnal cortisol pattern. For instance, some studies show that the HPA response hyperreactive with the CAR in chronically stressed populations (Hellhammer, Wüst, & Kudielka, 2009; Burke et al., 2005) while other studies show that basal cortisol levels are blunted in chronically stressed and burnt-out populations (Miller et al., 2007; Pruessner, Hellhammer, & Kirschbaum, 1999). This finding may occur within PTSD populations as well. Specifically, cortisol output of the HPA is increased by exposure to perceived stress although this blunted reactivity to stress is seen in those with PTSD diagnoses. It is speculated in the

research that the blunted HPA stress response may facilitate the development of PTSD (Miller et al., 2007). As these findings find effects in the blunted response of the HPA axis to a stressor and cross-talk between testosterone and cortisol occur during stressor exposure, it is highly recommended that future research collects hormone samples prior to and following an acute laboratory stress, if feasible (Josephs et al., 2017; Cobb et al., 2018).

Use of a different regression estimation technique than ordinary least squares variance estimation is recommended. For instance, use of multi-level regression (MLR; also known as mixed effect modeling) is advantageous as it can include more than one error variable in the regression as random effects – e.g., intercepts per study participant or intercepts per study participant per data collection period, while fixed effects remain very similarly calculated as is done in OLS. MLR also allows for hierarchal or nested data modeling. For example, within this sample, MLR could predict effects within the individual first responder at one level as well as effects for the entire group of local first responders. The random effect terms in MLR are capable of predicting outcome variables while accounting for multiple repeated measures within a sample. MLR does still assume that residuals of the analyses are normally distributed, so approaches to transforming predictor data values or excluding outliers would still need to be conducted in this dataset which demonstrated a violation of normality of the residuals.

The ability to operationalize stress variables further is available. Given the preponderance of stress research suggesting that perceived stress and objective measures of stressors are both significant to our understanding of allostatic load, the inclusion of objective measures of stressors (such as the critical incident call data explored supplementary within this study) would likely allow greater explanation of unique variance of clinical symptoms within diathesis-stress frameworks for psychopathology, regardless if hormone predictors are included or not. This study collected an objective measure of

stress consisting of number critical incidents (i.e., work calls in the field) to which the first responder responded over the three months prior to the data collection time point. Higher Alpha calls were predictive of lower basal cortisol levels, higher perceived stress, and lower resiliency ratings at baseline. Bravo calls, only the next step up in severity classification, predicted an increase in baseline resiliency. This reversal of effect on outcomes between Alpha and Bravo calls was also observed when predicting clinical outcomes at baseline. Higher Alpha calls experienced in the 3 months prior to baseline were predictive of higher PTSD symptoms, depression symptoms, and anxiety symptoms, but higher Bravo calls reversed one of those effects resulting in lower depression symptoms.

It is possible that the least severe category of calls (Alpha) is representative of any individual's encounter of daily stressors and work hassles – e.g., appraised as more common nuisance than threat. However, once calls increase in severity to the next level of medical emergency (Bravo), more skilled first response training may kick in to eliminate the effect of responding the call increasing a first responders perceived chronic stress. In modest evidence of this theory, the total incident call numbers in the three months prior to baseline was associated with less perceived stress (while Alpha calls were associated with were associated with more perceived stress). First responders may have the necessary training, social support, and innate resiliency to respond to a large number of calls without impacting chronic stress and thereby risking mental illness as long as calls are not predominantly of the Alpha classification. Future studies of this possible percentage of Alpha incidents as an environmental predictor for stress-linked psychopathology is indicated.

These main effects are encouraging for continued work modeling perceived stress variables, such as Cohen's self-reported PSS, with other quantitative data such as call data to approximate an exposure level to environmental stressors for an individual. Call data, such as was collected by this study, worked particularly well as every call for each first responder is catalogued and its severity classified according to the same guidelines for every first responder. This consistency of the data provides reliability between responders but also within responders across time.

Furthermore, opportunity to further operationalize the subjective perceived stress variable exists. An individual's total perceived stress score can be further broken down into 1.) a perceived stress rating for the individual at each collection timepoint, as well as, 2.) a deviation score of this collection time point's stress total from the mean of all perceived stress measures collected from the same individual throughout the study time frame. This second score is a deviation score to represent intra-individual perceived stress variability over time. Cohen's PSS was administered at each of the three data collection timepoints in this study although only baseline perceived stress was modeled in the hypothesized regressions. Combined use of these two variables of perceived stress (between-individual and within-individual effects) have been found to be significantly associated with risk predictions of metabolic dysregulation and diabetes (Aikens, Wallander, Bell, & Cole, 1992), emotional reactivity (Sliwinksi, Almeida, Smyth, & Stawski, 2009), PTSD (Josephs et al, 2017), and depression (Cobb et al., 2018). In fact, the dual hormone moderation of PTSD symptoms occurred when interacting with the between-subject stress variable only (Josephs et al., 2017), and dual hormone moderation of the depression symptoms occurred when interacting with the within-subject stress variable only (Cobb et al., 2018). Incorporating both concepts of perceived stress to various physiological stress variables (e.g., endocrine levels) and objective stress data (e.g., incident call data) is supported by

the stress research literature and will investigate capturing a greater proportion of uniquely explained variance for clinical psychopathologies.

Finally, future studies will also want to explore the differences in risk for a new cadet in the profession versus an experienced responder that has been in the field for longer. This design would investigate if different diatheses interact differently across the subsets of responders by years of experience within the service. While stratification of data is possible, it may be worthwhile to focus data recruitment and data collection on a single subset of responders (e.g., cadets prior to and during the first year of the job, or responders with 10-15 years of experience). The same study stratification would be possible for baseline severity of mental health symptoms as well. This aspect of revised study design would allow investigation of whether diatheses interact with stress difference for someone who already is experiencing a moderate to severe level of psychopathology (e.g., depression symptoms on a clinical screener) versus a responder who is reporting subclinical levels of psychopathology? In fact, we would expect that severity and duration of psychopathology is likely to be associated with changes to the HPA and HPG axes resulting in altered basal cortisol and testosterone levels. Subsetting models within responders by years on the force and presence of baseline psychopathology is a next step forward with multi-level regression modeling for longitudinal data for emergency first responders.

CONCLUSIONS

In conclusion, the present study aimed to generate new insights into hormone (specifically, cortisol and testosterone) moderated stress-linked mental health outcomes in first responders by modeling risk factors for psychopathology collected over a 6-month time frame. With a large number of comparisons corrected with a lowered adjusted Type

1 error probability for statistical significance and a small sample size, no hypothesized single and dual hormone effects were statistically supported. However, given the established literature on the single-effects of cortisol and testosterone on health and psychopathology, the stress-buffering factors of social support and resiliency, and the growing swath of dual hormone diathesis-stress studies, this study points out its statistical limitations and argues for continued dual hormone stress research into this chronically stressed population.

Appendices

APPENDIX A: STUDY DEMOGRAPHICS

1. What is your gender?
   O Female O Male O Transgender (F to M) O Transgender (M to F)

2. How old are you? _____ Years

3. Are you a military Veteran?
   O No O Yes

4. Which of the following best describes your current relationship status?
   O Single, not dating O Engaged to be married O Married but separated
   O Single, in casual relationship O Married, living with spouse/partner O Divorced
   O Single, in serious relationship O Married, geographically separated O Widowed

5. Do you currently live with your intimate/romantic partner?
   O No O Yes O Not applicable (not currently involved with partner)

6. Which of the following best describes your highest level of education?
   O some College, no degree O Associate Degree O Bachelor Degree
   O some Graduate School, no degree O Graduate Degree (please specify): _____

7. Are you Hispanic/Latino?
   O No O Yes (please specify below, select all that apply)

   O Cuban O Dominican O Mexican/Mexican-American O Puerto Rican
   O Spanish/Basque O Other (please specify): _____

8. To which racial group do you consider yourself belonging? (please select all that apply)
   O American-Indian or Alaska Native O Asian or Asian-American
   O Black or African-American O Native Hawaiian or other Pacific Islander
   O White or Caucasian O Other (please specify): _____

9. On average, how many hours per week have you worked in the last two (2) months?
   _____ hours

10. What is your current annual income (last 12 months)?
    O $0 - $14,999 O $15,000 - $29,999 O $30,000 - $44,999 O $45,000 - $59,999
    O $60,000 - $74,999 O $75,000 - $89,999 O $90,000 or higher

11. What is your current position?
    O EMT O Paramedic O Captain O Commander O Division Chief

O Associate Director O 911 Call Operator

12. How long have you worked at this position?
    O < 1 year O 1 to 3 years O 4 to 5 years O 6 to 10 years
    O 11 to 15 years O 16 to 20 years O >21 years

13. How long have you worked for ATCEMS?
    O < 1 year O 1 to 3 years O 4 to 5 years O 6 to 10 years
    O 11 to 15 years O 16 to 20 years O >21 years

14. On what date did you enter the training academy? _____ (MM/YYYY)

15. How long did your training academy last? _____(No. of weeks)

16. How many days of work did you miss in the past year for any reason other than planned vacation?
    O 0 O 1 O 2 O 3 O 4 O 5-9 O 10-14 O 15-19 O 20-24 O 25 or more

17. Are you currently receiving help for any concern from a mental health professional?

    O No O Yes

APPENDIX B: PERCEIVED STRESS MEASURE

Perceived Stress Scale (PSS)

Instructions: The questions in this scale ask you about your feelings and thoughts during the PAST MONTH. In each case, please indicate with a check how often you felt or thought a certain way.

___0=never ___1=almost never ___2=sometimes ___3= fairly often ___4=very often

1. In the last month, how often have you been upset because of something that happened unexpectedly? _____
2. In the last month, how often have you felt that you were unable to control the important things in your life? _____
3. In the last month, how often have you felt nervous and "stressed"? ____
4. In the last month, how often have you felt confident about your ability to handle your personal problems? ____
5. In the last month, how often have you felt that things were going your way? ____
6. In the last month, how often have you found that you could not cope with all the things that you had to do? ____
7. In the last month, how often have you been able to control irritations in your life? ___
8. In the last month, how often have you felt that you were on top of things? ___
9. In the last month, how often have you been angered because of things that were outside of your control? ____
10. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them? ____

APPENDIX C: SOCIAL SUPPORT MEASURE

Interpersonal Support Evaluation List (ISEL)

Instructions: This scale is made up of a list of statements each of which may or may not be true about you. For each statement, circle "definitely true" if you are sure it is true about you and "probably true" if you think it is true but are not certain. Similarly, you should circle "definitely false" if you are sure the statement is false and "probably false" if you think it is false but are not certain.

1. If I wanted to go on a trip for a day (for example, to the country or mountains), I would have a hard time finding someone to go with me.
   1. definitely false 2. probably false 3. probably true 4. definitely true
2. I feel that there is no one I can share my most private worries and fears with.
   1. definitely false 2. probably false 3. probably true 4. definitely true
3. If I were sick, I could easily find someone to help me with my daily chores.
   1. definitely false 2. probably false 3. probably true 4. definitely true
4. There is someone I can turn to for advice about handling problems with my family.
   1. definitely false 2. probably false 3. probably true 4. definitely true
5. If I decide one afternoon that I would like to go to a movie that evening, I could easily find someone to go with me.
   1. definitely false 2. probably false 3. probably true 4. definitely true
6. When I need suggestions on how to deal with a personal problem, I know someone I can turn to.
   1. definitely false 2. probably false 3. probably true 4. definitely true
7. I don't often get invited to do things with others.
   1. definitely false 2. probably false 3. probably true 4. definitely true
8. If I had to go out of town for a few weeks, it would be difficult to find someone who would look after my house or apartment (the plants, pets, garden, etc.).
   1. definitely false 2. probably false 3. probably true 4. definitely true
9. If I wanted to have lunch with someone, I could easily find someone to join me.
   1. definitely false 2. probably false 3. probably true 4. definitely true
10. If I was stranded 10 miles from home, there is someone I could call who could come and get me.
    1. definitely false 2. probably false 3. probably true 4. definitely true
11. If a family crisis arose, it would be difficult to find someone who could give me good advice about how to handle it.
    1. definitely false 2. probably false 3. probably true 4. definitely true
12. If I needed some help in moving to a new house or apartment, I would have a hard time finding someone to help me.
    1. definitely false 2. probably false 3. probably true 4. definitely true

APPENDIX D: RESILIENCY MEASURE

Brief Resiliency Scale

Instructions: Please indicate the extent to which you agree with each of the following statements by using the following scale:

1 = strongly disagree 2 = disagree 3 = neutral 4 = agree 5 = strongly agree

1. I tend to bounce back quickly after hard times.
   1 2 3 4 5
2. I have a hard time making it through stressful events.
   1 2 3 4 5
3. It does not take me long to recover form a stressful event.
   1 2 3 4 5
4. It is hard for me to snap back when something bad happens.
   1 2 3 4 5
5. I usually come through difficult times with little trouble.
   1 2 3 4 5
6. I tend to take a long time to get over setbacks in my life.
   1 2 3 4 5

APPENDIX E: POST-TRAUMATIC STRESS DISORDER SYMPTOM INVENTORY

PTSD Checklist – Civilian Version (PCL-C)

Instructions: Below is a list of problems and complaints that veterans sometimes have in response to stressful life experiences. Please read each question carefully and circle in the box to indicate **how much** you have been bothered by that problem in the LAST MONTH.

1 = Not at all 2 = A little bit 3 = Moderately 4 = Quite a bit 5 = Extremely

	Not at all	A little bit	Moderately	Quite a bit	Extremely
1. Repeated, disturbing *memories, thoughts, or images* of a stressful experience from the past?	1	2	3	4	5
2. Repeated, disturbing *dreams* of a stressful experience from the past?	1	2	3	4	5
3. Suddenly *acting* or *feeling* as if a stressful experience *were happening* again (as if you were reliving it)?	1	2	3	4	5
4. Feeling *very upset* when *something reminded* you of a stressful experience from the past?	1	2	3	4	5
5. Having physical reactions (e.g., heart pounding, trouble breathing, or sweating) when something reminded you of a stressful experience from the past?	1	2	3	4	5
6. Avoid thinking about or talking about a stressful experience from the past or avoid having feelings related to it?	1	2	3	4	5

7. Avoid activities or situations because they remind you of a stressful experience from the past?	1	2	3	4	5
8. Trouble remembering important parts of a stressful experience from the past?	1	2	3	4	5
9. Loss of interest in things that you used to enjoy?	1	2	3	4	5
10. Feeling distant or cut off from other people?	1	2	3	4	5
11. Feeling emotionally numb or being unable to have loving feelings for those close to you?	1	2	3	4	5
12. Feeling as if your future will somehow be cut short?	1	2	3	4	5
13. Trouble falling or staying asleep?	1	2	3	4	5
14. Feeling irritable or having angry outbursts?	1	2	3	4	5
15. Having difficulty concentrating?	1	2	3	4	5
16. Being “super alert” or watchful on guard?	1	2	3	4	5
17. Feeling jumpy or easily startled?	1	2	3	4	5

APPENDIX F: DEPRESSION SYMPTOM INVENTORY

Center for Epidemiological Studies Depression Scale – Short Form (CESD-10)
Instructions: Below is a list of the ways you might have felt or behaved. Please tell me **how often** you have felt this way during the PAST WEEK, INCLUDING TODAY. Circle your response in the appropriate column for each question.
0 = Rarely or none of the time (less than 1 day)
1 = Some or a little of the time (1-2 days)
2 = Occasionally or a moderate amount of the time (3-4 days)
3 = Most or all of the time (5-7 days)

	Rarely or none of the time (<1 day)	**Some or a little of the time** (1-2 days)	**Occasionally or a moderate amount of the time** (3-4 days)	**Most or all of the time** (5-7 days)
1. I was bothered by things that usually don't bother me.	0	1	2	3
2. I had trouble keeping my mind on what I was doing.	0	1	2	3
3. I felt depressed.	0	1	2	3
4. I felt hopeful about the future.	0	1	2	3
5. I felt fearful.	0	1	2	3
6. My sleep was restless.	0	1	2	3
7. I was happy.	0	1	2	3
8. I felt lonely.	0	1	2	3
9. I felt that everything I did was an effort.	0	1	2	3
10. I could not get "going."	0	1	2	3

APPENDIX G: ALCOHOL USE MEASURE

Modified Daily Drinking Questionnaire (modified DDQ)

Instructions: For the following questions, please think about your drinking behavior since the last assessment. If you have not consumed ANY alcohol at all since the last assessment, please indicate below.

A. In a typical 3-month period, there are about 13 weeks and so there are usually 13 Mondays, 13 Tuesdays, and so on. For each day of the week, please write down the number of weeks (out of 13) that you drank any alcohol during the past 3 months. For example, if you never drank on any Tuesday, you would write "0" on the line for Tuesdays. If you drank on every Friday in that 13-week period, you would write "13" on the line for Fridays.

   Monday: _____ weeks
   Tuesday: _____ weeks
   Wednesday: _____ weeks
   Thursday: _____ weeks
   Friday: _____ weeks
   Saturday: _____ weeks
   Sunday: _____ weeks

B. Thinking about only the days you consumed alcohol during the same 13-week period indicated above, please write the average number of STANDARD drinks you consumed on each of those days. For example, if you drank on 3 Saturdays you would write down the average number of STANDARD drinks you had on those 3 days. Please write "0" for any days of the week that you did not drink in the last three months (i.e., any days you answered "0" for above).

   1 Standard Drink = 12 ounces of beer, 1 shot of liquor (straight or in mixed drink), or 5 ounces of wine

   Monday: _____ average standard drinks
   Tuesday: _____ average standard drinks
   Wednesday: _____ average standard drinks
   Thursday: _____ average standard drinks
   Friday: _____ average standard drinks
   Saturday: _____ average standard drinks
   Sunday: _____ average standard drinks

C. If you marked zero for all seven days in an average week above, was it because (*check one*):

___You never drink alcohol.
___You rarely drink alcohol (i.e., alcohol use is not typical for you).
___You typically drink alcohol but did not drink since the last assessment.

If you have not consumed ANY alcohol since the last assessment, please write a "0" on each line for questions 1-4 and answer questions 5-8 below.

1. Since the last assessment, how many times did you get a little high, lightheaded, or "buzzed" (but not drunk)?

   _____ times
2. Since the last assessment, how many times did you get drunk (more than just a little high, lightheaded, or "buzzed")?

   _____ times
3. Since the last assessment, how many times did you have:

   MEN: 5 or more drinks in one sitting? _____ times

   WOMEN: 4 or more drinks in one sitting? _____ times
4. Since the last assessment, what is the MOST number of standard drinks that you consumed in one sitting?

   _____drinks
5. How many standard drinks would you need to consume over a 30-minute period to feel a little high, lightheaded, or buzzed?

   _____ drinks
6. How many standard drinks would you need to consume over a 30-minute period to feel drunk?

   _____ drinks
7. Do you feel you have a high tolerance to alcohol?

   _____ No

   _____ Yes

   _____ Uncertain
8. Which of these is closest to your experience? Compared to other people does it takes:

   _____ more alcohol for you to become impaired

   _____ less alcohol for you to become impaired

   _____ about the same amount of alcohol for you to become impaired

APPENDIX H: SLEEP QUALITY MEASURE

Pittsburgh Sleep Quality Inventory (PSQI)

Instructions: The following questions relate to your usual sleep habits during the PAST MONTH only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month.

1. When have you usually gone to bed? _____
2. How long (in minutes) has it taken you to fall asleep each night? _____
3. When have you usually gotten up in the morning? _____
4. How many hours of actual sleep do you get at night? (This may be different than the number of hours you spend in bed.) _____

Mark an X in the appropriate column for each question.

5. During the past month, how often have you had trouble sleeping because you…	Not during the past month (0)	Less than once a week (1)	Once or twice a week (2)	Three of more times a week (3)
A. Cannot get to sleep within 30 minutes.				
B. Wake up in the middle of the night or early morning.				
C. Have to get up to use the bathroom.				
D. Cannot breathe comfortably.				
E. Cough or snore loudly.				
F. Feel too cold.				
G. Feel too hot.				
H. Have bad dreams.				
I. Have pain				
J. Other reason(s)				
6. During the past month, how often have you taken medicine (prescribed or "over the counter") to help you sleep?				
7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?				
8. During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done?				
	Very good (0)	Fairly good (1)	Fairly bad (2)	Very bad (3)
9. During the past month, how would you rate your sleep quality overall?				

APPENDIX I : FULL REGRESSION TABLES

Regression tables for all statistical models are listed below by predictor variable. For example, all cortisol-only regression models are listed for predicted PTSD, depression, anxiety, weekly alcohol consumption, and sleep quality within

Cortisol-Only Models

	PTSD Symptoms after 3 Months						PTSD Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	6.19	2.50	2.48	0.015*	120	0.22	5.82	3.32	1.75	0.084	78	0.19
Cortisol (C)	-0.43	2.10	-0.21	0.837	120	0.02	1.11	2.85	0.39	0.697	78	0.04
Perceived Stress (PS)	0.13	0.18	0.72	0.472	120	0.07	0.56	0.26	2.13	0.036*	78	0.23
Baseline PTSD	0.70	0.10	6.85	<0.001***	120	0.53	0.51	0.15	3.28	0.002**	78	0.35
C * PS	0.11	0.15	0.71	0.479	120	0.06	0.02	0.18	0.12	0.908	78	0.01
	Depression Symptoms after 3 Months						Depression Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	2.41	0.89	2.70	0.008**	110	0.25	-0.27	1.29	-0.21	0.837	76	0.02
Cortisol (C)	-0.86	0.88	-0.98	0.331	110	0.09	0.97	1.25	0.78	0.438	76	0.09
Perceived Stress (PS)	0.18	0.08	2.22	0.028*	110	0.21	0.53	0.12	4.57	<0.001***	76	0.46
Baseline Depression	0.54	0.10	5.26	<0.001***	110	0.45	0.25	0.15	1.70	0.094	76	0.19
C * PS	0.11	0.06	1.75	0.084	110	0.16	-0.02	0.08	-0.28	0.778	76	0.03
	Anxiety Symptoms after 3 Months						Anxiety Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	0.53	1.25	0.43	0.669	122	0.04	-3.10	1.92	-1.62	0.109	88	0.17
Cortisol (C)	-0.72	1.18	-0.61	0.544	122	0.05	-0.66	1.82	-0.36	0.718	88	0.04
Perceived Stress (PS)	0.17	0.09	1.84	0.068	122	0.16	0.47	0.15	3.14	0.002**	88	0.32
Baseline Anxiety	0.61	0.10	6.28	<0.001***	122	0.49	0.65	0.15	4.40	<0.001***	88	0.42
C * PS	0.12	0.08	1.45	0.150	122	0.13	0.10	0.12	0.88	0.379	88	0.09
	Weekly Alcoholic Drinks after 3 Months						Weekly Alcoholic Drinks after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	12.14	5.13	2.37	0.020*	108	0.22	8.36	2.85	2.93	0.004**	79	0.31
Cortisol (C)	-3.36	4.65	-0.72	0.472	108	0.07	1.25	2.67	0.47	0.642	79	0.05

Perceived Stress (PS)	-0.12	0.30	-0.41	0.685	108	0.04	-0.13	0.17	-0.75	0.457	79	0.08
Baseline Alcohol/Wk	0.58	0.17	3.47	<0.001***	108	0.32	1.09	0.14	7.92	<0.001***	79	0.67
C * PS	0.19	0.33	0.59	0.557	108	0.06	-0.09	0.17	-0.49	0.627	79	0.05
	Sleep Quality after 3 Months						Sleep Quality after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	1.53	0.87	1.75	0.084	88	0.18	1.34	1.11	1.21	0.233	62	0.15
Cortisol (C)	-0.76	0.77	-0.99	0.323	88	0.11	-1.09	1.05	-1.04	0.304	62	0.13
Perceived Stress (PS)	<0.01	0.06	0.05	0.963	88	0.01	0.06	0.08	0.76	0.451	62	0.10
Baseline Sleep Quality	0.78	0.09	8.39	<0.001***	88	0.67	0.73	0.12	6.13	<0.001***	62	0.61
C * PS	0.08	0.05	1.52	0.133	88	0.16	0.08	0.07	1.17	0.247	62	0.15

Note. Effect sizes (*r)* were calculated using the formula $\sqrt{(t^2 / (t^2 + df))}$; p * <.05; ** $p < .01$, *** $p < .001$

Testosterone-Only Models

	PTSD Symptoms after 3 Months						PTSD Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	5.60	2.56	2.19	0.030*	120	0.20	5.22	3.31	1.58	0.119	78	0.18
Testosterone (T)	-0.21	2.23	-0.09	0.925	120	0.01	0.49	3.18	0.15	0.878	78	0.02
Perceived Stress (PS)	0.13	0.18	0.74	0.458	120	0.07	0.57	0.27	2.11	0.038*	78	0.23
Baseline PTSD	0.73	0.11	6.58	<0.001***	120	0.51	0.53	0.16	3.33	0.001**	78	0.35
T * PS	<0.01	0.11	-0.04	0.968	120	<0.01	-0.09	0.17	-0.52	0.608	78	0.06
	Depression Symptoms after 3 Months						Depression Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	2.06	0.90	2.30	0.023*	110	0.21	-0.20	1.27	-0.16	0.875	76	0.02
Testosterone (T)	1.33	0.90	1.47	0.144	110	0.14	0.99	1.30	0.77	0.445	76	0.09
Perceived Stress (PS)	0.18	0.08	2.31	0.023*	110	0.22	0.51	0.11	4.52	<0.001	76	0.46
Baseline Depression	0.59	0.11	5.52	<0.001***	110	0.47	0.28	0.14	1.96	0.054	76	0.22
T * PS	-0.06	0.04	-1.33	0.188	110	0.13	-0.04	0.07	-0.61	0.542	76	0.07
	Anxiety Symptoms after 3 Months						Anxiety Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	0.14	1.24	0.11	0.913	122	0.01	-4.03	1.89	-2.13	0.036*	88	0.22
Testosterone (T)	1.83	1.28	1.43	0.155	122	0.13	1.72	1.75	0.99	0.327	88	0.10

Perceived Stress (PS)	0.22	0.09	2.39	0.018*	122	0.21	0.51	0.15	3.53	<0.001***	88	0.35
Baseline Anxiety	0.59	0.10	5.80	<0.001***	122	0.46	0.71	0.15	4.64	<0.001***	88	0.44
T * PS	-0.06	0.06	-0.98	0.330	122	0.09	-0.13	0.08	-1.55	0.124	88	0.16
	Weekly Alcoholic Drinks after 3 Months						Weekly Alcoholic Drinks after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	10.87	5.12	2.12	0.036*	108	0.20	7.69	2.76	2.78	0.007**	79	0.30
Testosterone (T)	4.71	5.28	0.89	0.374	108	0.09	6.36	2.96	2.15	0.035*	79	0.24
Perceived Stress (PS)	-0.04	0.29	-0.15	0.878	108	0.01	-0.08	0.17	-0.47	0.641	79	0.05
Baseline Alcohol/Wk	0.61	0.17	3.56	<0.001***	108	0.32	1.08	0.13	7.99	<0.001***	79	0.67
T * PS	-0.24	0.25	-0.96	0.341	108	0.09	-0.31	0.16	-2.00	0.049*	79	0.22
	Sleep Quality after 3 Months						Sleep Quality after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	1.33	0.87	1.53	0.130	88	0.16	0.86	1.06	0.82	0.417	62	0.10
Testosterone (T)	0.43	0.92	0.47	0.642	88	0.05	0.84	1.22	0.69	0.492	62	0.09
Perceived Stress (PS)	0.02	0.05	0.38	0.708	88	0.04	0.11	0.07	1.59	0.117	62	0.20
Baseline Sleep Quality	0.79	0.09	8.45	<0.001***	88	0.67	0.69	0.11	6.03	<0.001	62	0.61
T * PS	<0.01	0.04	0.06	0.952	88	0.01	-0.08	0.06	-1.31	0.195	62	0.16

Note. Effect sizes (*r)* were calculated using the formula $\sqrt{(t^2 / (t^2 + df))}$; p * <.05; ** $p < .01$, *** $p < .001$

Dual Hormone (Cortisol and Testosterone) Models

	PTSD Symptoms after 3 Months						PTSD Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	5.10	2.79	1.83	0.071	116	0.17	5.65	3.56	1.59	0.116	74	0.18
Testosterone (T)	-0.89	2.74	-0.32	0.747	116	0.03	0.69	3.89	0.18	0.860	74	0.02
Cortisol (C)	-0.33	2.23	-0.15	0.884	116	0.01	0.37	3.44	0.11	0.914	74	0.01
Perceived Stress (PS)	0.14	0.19	0.75	0.454	116	0.07	0.51	0.29	1.76	0.083	74	0.20
Baseline PTSD	0.74	0.11	6.62	<0.001***	116	0.52	0.54	0.16	3.31	0.001**	74	0.36
T * C	0.83	2.58	0.32	0.749	116	0.03	-0.15	3.27	-0.05	0.963	74	0.01
T * PS	0.05	0.15	0.35	0.725	116	0.03	-0.14	0.22	-0.62	0.536	74	0.07
C * PS	0.09	0.16	0.56	0.579	116	0.05	0.10	0.22	0.45	0.657	74	0.05
T * C * PS	-0.14	0.19	-0.72	0.471	116	0.07	0.02	0.19	0.11	0.910	74	0.01

	Depression Symptoms after 3 Months						Depression Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	2.18	0.96	2.28	0.025*	106	0.22	-0.01	1.38	-0.01	0.995	72	<0.01
Testosterone (T)	1.52	1.09	1.39	0.168	106	0.13	0.93	1.53	0.61	0.546	72	0.07
Cortisol (C)	-1.32	0.94	-1.41	0.162	106	0.14	1.02	1.45	0.70	0.485	72	0.08
Perceived Stress (PS)	0.18	0.08	2.15	0.034*	106	0.20	0.51	0.12	4.23	<0.001***	72	0.45
Baseline Depression	0.59	0.11	5.54	<0.001***	106	0.47	0.25	0.15	1.68	0.098	72	0.19
T * C	0.02	1.02	0.02	0.988	106	<0.01	-0.96	1.36	-0.71	0.481	72	0.08
T * PS	-0.06	0.06	-0.93	0.353	106	0.09	-0.04	0.08	-0.54	0.591	72	0.06
C * PS	0.13	0.07	1.95	0.053	106	0.19	-0.03	0.09	-0.29	0.773	72	0.03
T * C * PS	-0.05	0.08	-0.63	0.529	106	0.06	0.07	0.08	0.80	0.427	72	0.09
	Anxiety Symptoms after 3 Months						Anxiety Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	1.11	1.32	0.84	0.402	118	0.08	-3.57	1.96	-1.83	0.072	84	0.20
Testosterone (T)	3.37	1.54	2.18	0.031*	118	0.20	2.24	2.05	1.10	0.277	84	0.12
Cortisol (C)	-1.36	1.25	-1.09	0.279	118	0.10	-2.01	2.06	-0.97	0.333	84	0.11
Perceived Stress (PS)	0.14	0.10	1.44	0.152	118	0.13	0.46	0.15	3.07	0.003**	84	0.32
Baseline Anxiety	0.59	0.10	5.71	<0.001***	118	0.47	0.74	0.16	4.50	<0.001***	84	0.44
T * C	-1.97	1.47	-1.34	0.184	118	0.12	0.30	2.03	0.15	0.884	84	0.02
T * PS	-0.17	0.09	-2.00	0.048*	118	0.18	-0.18	0.10	-1.77	0.080	84	0.19
C * PS	0.17	0.09	1.87	0.064	118	0.17	0.20	0.13	1.53	0.130	84	0.16
T * C * PS	0.15	0.11	1.38	0.170	118	0.13	0.03	0.12	0.21	0.837	84	0.02
	Weekly Alcoholic Drinks after 3 Months						Weekly Alcoholic Drinks after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	11.21	5.85	1.92	0.058	104	0.18	7.67	2.97	2.58	0.012*	75	0.29
Testosterone (T)	6.46	6.51	0.99	0.323	104	0.10	7.19	3.49	2.06	0.043*	75	0.23
Cortisol (C)	-5.06	5.07	-1.00	0.321	104	0.10	-1.37	3.02	-0.45	0.652	75	0.05
Perceived Stress (PS)	-0.05	0.35	-0.15	0.880	104	0.01	-0.05	0.18	-0.27	0.787	75	0.03
Baseline Alcohol/Wk	0.63	0.18	3.46	<0.001***	104	0.32	1.05	0.14	7.50	<0.001***	75	0.65
T * C	-0.73	6.04	-0.12	0.904	104	0.01	0.44	2.97	0.15	0.883	75	0.02
T * PS	-0.28	0.37	-0.75	0.452	104	0.07	-0.34	0.18	-1.89	0.063	75	0.21
C * PS	0.29	0.37	0.77	0.443	104	0.08	0.05	0.19	0.27	0.788	75	0.03
T * C * PS	-0.07	0.46	-0.16	0.874	104	0.02	-0.11	0.18	-0.59	0.560	75	0.07
	Sleep Quality after 3 Months						Sleep Quality after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*

Intercept	1.59	0.94	1.68	0.096	84	0.18	1.41	1.20	1.17	0.246	58	0.15
Testosterone (T)	0.86	1.14	0.76	0.452	84	0.08	1.28	1.55	0.83	0.410	58	0.11
Cortisol (C)	-0.62	0.80	-0.78	0.438	84	0.08	-2.09	1.16	-1.79	0.078	58	0.23
Perceived Stress (PS)	<0.01	0.06	-0.03	0.981	84	<0.01	0.04	0.08	0.53	0.596	58	0.07
Baseline Sleep Quality	0.79	0.10	8.08	<0.001***	84	0.66	0.73	0.12	5.96	<0.001***	58	0.62
T * C	-0.52	0.96	-0.54	0.589	84	0.06	0.43	1.34	0.32	0.751	58	0.04
T * PS	-0.02	0.06	-0.35	0.731	84	0.04	-0.13	0.09	-1.39	0.171	58	0.18
C * PS	0.07	0.06	1.20	0.234	84	0.13	0.17	0.08	2.09	0.041*	58	0.26
T * C * PS	0.02	0.07	0.28	0.783	84	0.03	-0.01	0.10	-0.08	0.934	58	0.01

Note. Effect sizes (*r)* were calculated using the formula $\sqrt{(t^2 / (t^2 + df))}$; p * <.05; ** p < .01, *** p < .001

Cortisol and Social Support Models

	PTSD Symptoms after 3 Months						PTSD Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	9.93	14.26	0.70	0.488	114	0.07	10.29	18.40	0.56	0.578	74	0.06
Cortisol (C)	-2.67	14.74	-0.18	0.857	114	0.02	-6.01	18.39	-0.33	0.745	74	0.04
Perceived Stress (PS)	0.17	0.79	0.22	0.829	114	0.02	1.30	1.10	1.18	0.241	74	0.14
Social Support (SS)	-0.07	0.34	-0.20	0.845	114	0.02	-0.02	0.44	-0.06	0.956	74	0.01
Baseline PTSD	0.67	0.11	6.19	<0.001***	114	0.50	0.39	0.17	2.35	0.022*	74	0.26
C * PS	0.64	0.79	0.80	0.423	114	0.08	0.90	1.00	0.90	0.369	74	0.10
C * SS	0.08	0.35	0.22	0.824	114	0.02	0.21	0.44	0.49	0.628	74	0.06
PS * SS	<0.01	0.02	-0.06	0.955	114	0.01	-0.02	0.03	-0.77	0.446	74	0.09
C * PS * SS	-0.02	0.02	-0.76	0.451	114	0.07	-0.03	0.03	-1.05	0.296	74	0.12
	Depression Symptoms after 3 Months						Depression Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	10.17	5.84	1.74	0.084	104	0.17	2.84	7.59	0.37	0.710	72	0.04
Cortisol (C)	13.32	6.24	2.14	0.035*	104	0.20	11.46	7.73	1.48	0.142	72	0.17
Perceived Stress (PS)	0.14	0.33	0.42	0.677	104	0.04	1.04	0.45	2.31	0.024*	72	0.26
Social Support (SS)	-0.16	0.14	-1.18	0.239	104	0.12	-0.04	0.18	-0.24	0.814	72	0.03
Baseline Depression	0.45	0.12	3.90	<0.001***	104	0.36	0.04	0.16	0.23	0.819	72	0.03
C * PS	-0.50	0.33	-1.51	0.135	104	0.15	-0.72	0.44	-1.65	0.103	72	0.19
C * SS	-0.32	0.15	-2.24	0.027*	104	0.21	-0.25	0.19	-1.33	0.187	72	0.16

PS * SS	<0.01	0.01	0.05	0.958	104	0.01	-0.01	0.01	-1.17	0.246	72	0.14
C * PS * SS	0.01	0.01	1.64	0.105	104	0.16	0.02	0.01	1.53	0.131	72	0.18
	Anxiety Symptoms after 3 Months						Anxiety Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	5.25	8.14	0.64	0.521	116	0.06	-12.75	11.27	-1.13	0.261	84	0.12
Cortisol (C)	-10.64	8.19	-1.30	0.196	116	0.12	-6.73	10.92	-0.62	0.540	84	0.07
Perceived Stress (PS)	-0.12	0.45	-0.26	0.797	116	0.02	1.49	0.67	2.22	0.029*	84	0.24
Social Support (SS)	-0.11	0.19	-0.57	0.569	116	0.05	0.27	0.27	1.02	0.312	84	0.11
Baseline Anxiety	0.64	0.10	6.49	<0.001***	116	0.52	0.54	0.15	3.51	<0.001***	84	0.36
C * PS	1.44	0.44	3.29	0.001**	116	0.29	0.98	0.62	1.58	0.118	84	0.17
C * SS	0.29	0.19	1.50	0.136	116	0.14	0.19	0.26	0.73	0.469	84	0.08
PS * SS	0.01	0.01	0.57	0.569	116	0.05	-0.03	0.02	-1.75	0.084	84	0.19
C * PS * SS	-0.04	0.01	-3.45	<0.001***	116	0.31	-0.03	0.02	-1.71	0.090	84	0.18
	Weekly Alcoholic Drinks after 3 Months						Weekly Alcoholic Drinks after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	24.92	35.59	0.70	0.485	102	0.07	-0.07	17.82	<0.01	0.997	75	<0.01
Cortisol (C)	55.84	37.14	1.50	0.136	102	0.15	16.41	17.38	0.94	0.348	75	0.11
Perceived Stress (PS)	-0.40	1.77	-0.23	0.821	102	0.02	0.57	0.95	0.60	0.548	75	0.07
Social Support (SS)	-0.29	0.84	-0.34	0.732	102	0.03	0.22	0.43	0.51	0.609	75	0.06
Baseline Alcohol/Wk	0.61	0.17	3.50	<0.001***	102	0.33	1.11	0.14	7.86	<0.001***	75	0.67
C * PS	-2.30	1.88	-1.22	0.225	102	0.12	-1.55	0.96	-1.61	0.111	75	0.18
C * SS	-1.38	0.87	-1.59	0.114	102	0.16	-0.40	0.42	-0.97	0.337	75	0.11
PS * SS	<0.01	0.04	0.11	0.914	102	0.01	-0.02	0.02	-0.75	0.455	75	0.09
C * PS * SS	0.06	0.05	1.21	0.229	102	0.12	0.04	0.02	1.64	0.105	75	0.19
	Sleep Quality after 3 Months						Sleep Quality after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	7.07	5.64	1.25	0.214	83	0.14	13.36	7.33	1.82	0.073	58	0.23
Cortisol (C)	2.84	5.76	0.49	0.623	83	0.05	0.91	7.04	0.13	0.897	58	0.02
Perceived Stress (PS)	-0.13	0.29	-0.45	0.654	83	0.05	-0.69	0.42	-1.65	0.105	58	0.21
Social Support (SS)	-0.12	0.13	-0.91	0.367	83	0.10	-0.30	0.17	-1.72	0.092	58	0.22
Baseline Sleep Quality	0.76	0.10	7.83	<0.001***	83	0.65	0.69	0.12	5.64	<0.001***	58	0.60
C * PS	-0.11	0.31	-0.37	0.711	83	0.04	0.20	0.42	0.49	0.629	58	0.06
C * SS	-0.09	0.14	-0.64	0.526	83	0.07	-0.04	0.17	-0.25	0.801	58	0.03
PS * SS	<0.01	0.01	0.35	0.725	83	0.04	0.02	0.01	1.82	0.074	58	0.23
C * PS * SS	<0.01	0.01	0.61	0.547	83	0.07	<0.01	0.01	-0.34	0.738	58	0.04

Note. Effect sizes (r) were calculated using the formula $\sqrt{(t^2 / (t^2 + df))}$; p * <.05; ** p < .01, *** p < .001

Testosterone and Social Support Models

	PTSD Symptoms after 3 Months						PTSD Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	8.02	14.24	0.56	0.574	114	0.05	3.43	17.85	0.19	0.848	74	0.02
Testosterone (T)	-9.85	15.56	-0.63	0.528	114	0.06	0.29	20.39	0.01	0.989	74	<0.01
Perceived Stress (PS)	0.48	0.78	0.62	0.537	114	0.06	1.98	1.01	1.96	0.054	74	0.22
Social Support (SS)	-0.02	0.34	-0.05	0.963	114	<0.01	0.15	0.43	0.36	0.719	74	0.04
Baseline PTSD	0.66	0.12	5.63	<0.001***	114	0.47	0.37	0.17	2.16	0.034*	74	0.24
T * PS	0.59	0.64	0.92	0.359	114	0.09	0.43	1.06	0.41	0.686	74	0.05
T * SS	0.27	0.37	0.73	0.464	114	0.07	0.05	0.50	0.10	0.921	74	0.01
PS * SS	-0.01	0.02	-0.45	0.655	114	0.04	-0.04	0.02	-1.54	0.128	74	0.18
T * PS * SS	-0.02	0.02	-1.03	0.306	114	0.10	-0.02	0.03	-0.61	0.547	74	0.07
	Depression Symptoms after 3 Months						Depression Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	6.97	5.87	1.19	0.238	104	0.12	4.05	7.57	0.53	0.595	72	0.06
Testosterone (T)	6.28	6.35	0.99	0.326	104	0.10	4.37	8.53	0.51	0.610	72	0.06
Perceived Stress (PS)	0.20	0.32	0.61	0.545	104	0.06	0.76	0.41	1.86	0.068	72	0.21
Social Support (SS)	-0.10	0.14	-0.73	0.467	104	0.07	-0.08	0.18	-0.41	0.680	72	0.05
Baseline Depression	0.53	0.12	4.49	<0.001***	104	0.40	0.15	0.15	0.96	0.339	72	0.11
T * PS	-0.37	0.26	-1.41	0.162	104	0.14	-0.26	0.44	-0.60	0.553	72	0.07
T * SS	-0.14	0.15	-0.93	0.353	104	0.09	-0.09	0.21	-0.43	0.668	72	0.05
PS * SS	<0.01	0.01	-0.09	0.928	104	0.01	-0.01	0.01	-0.69	0.491	72	0.08
T * PS * SS	0.01	0.01	1.33	0.187	104	0.13	0.01	0.01	0.51	0.613	72	0.06
	Anxiety Symptoms after 3 Months						Anxiety Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	-1.74	8.51	-0.21	0.838	116	0.02	-23.55	10.71	-2.20	0.031*	84	0.23
Testosterone (T)	-14.78	9.08	-1.63	0.107	116	0.15	8.46	10.89	0.78	0.439	84	0.08
Perceived Stress (PS)	0.45	0.46	0.99	0.327	116	0.09	2.35	0.58	4.03	<0.001***	84	0.40
Social Support (SS)	0.05	0.20	0.26	0.795	116	0.02	0.53	0.26	2.08	0.041*	84	0.22

Baseline Anxiety	0.54	0.11	5.02	<0.001***	116	0.42	0.55	0.15	3.55	<0.001***	84	0.36
T * PS	0.64	0.37	1.72	0.088	116	0.16	-0.44	0.45	-1.00	0.323	84	0.11
T * SS	0.41	0.22	1.90	0.060	116	0.17	-0.17	0.26	-0.66	0.512	84	0.07
PS * SS	-0.01	0.01	-0.52	0.605	116	0.05	-0.05	0.01	-3.44	<0.001***	84	0.35
T * PS * SS	-0.02	0.01	-1.83	0.071	116	0.17	0.01	0.01	0.67	0.502	84	0.07
	Weekly Alcoholic Drinks after 3 Months						Weekly Alcoholic Drinks after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	29.77	35.47	0.84	0.403	102	0.08	9.90	16.92	0.59	0.560	75	0.07
Testosterone (T)	-28.95	37.75	-0.77	0.445	102	0.08	15.99	20.12	0.79	0.429	75	0.09
Perceived Stress (PS)	-0.93	1.75	-0.53	0.596	102	0.05	-0.17	0.86	-0.20	0.840	75	0.02
Social Support (SS)	-0.46	0.84	-0.55	0.585	102	0.05	-0.06	0.40	-0.14	0.886	75	0.02
Baseline Alcohol/Wk	0.65	0.18	3.57	<0.001***	102	0.33	1.09	0.14	7.76	<0.001***	75	0.67
T * PS	0.66	1.49	0.44	0.658	102	0.04	-0.03	1.01	-0.03	0.974	75	<0.01
T * SS	0.78	0.92	0.85	0.398	102	0.08	-0.19	0.49	-0.38	0.702	75	0.04
PS * SS	0.02	0.04	0.51	0.614	102	0.05	<0.01	0.02	0.10	0.925	75	0.01
T * PS * SS	-0.02	0.04	-0.45	0.657	102	0.04	-0.01	0.03	-0.43	0.672	75	0.05
	Sleep Quality after 3 Months						Sleep Quality after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	6.78	5.49	1.24	0.220	83	0.13	11.86	6.74	1.76	0.084	58	0.22
Testosterone (T)	-7.95	6.45	-1.23	0.222	83	0.13	-9.55	8.06	-1.19	0.241	58	0.15
Perceived Stress (PS)	-0.16	0.27	-0.58	0.561	83	0.06	-0.40	0.33	-1.23	0.223	58	0.16
Social Support (SS)	-0.13	0.13	-0.98	0.329	83	0.11	-0.27	0.16	-1.70	0.095	58	0.22
Baseline Sleep Quality	0.80	0.09	8.39	0.001***	83	0.68	0.66	0.12	5.51	<0.001***	58	0.59
T * PS	0.17	0.26	0.65	0.518	83	0.07	0.48	0.40	1.18	0.242	58	0.15
T * SS	0.18	0.16	1.14	0.260	83	0.12	0.28	0.21	1.38	0.174	58	0.18
PS * SS	<0.01	0.01	0.58	0.567	83	0.06	0.01	0.01	1.57	0.121	58	0.20
T * PS * SS	<0.01	0.01	-0.32	0.753	83	0.03	-0.02	0.01	-1.41	0.163	58	0.18

Note. Effect sizes (*r)* were calculated using the formula $\sqrt{(t^2 / (t^2 + df))}$; p * <.05; ** $p < .01$, *** $p < .001$

Cortisol and Resiliency Models

	PTSD Symptoms after 3 Months						PTSD Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*

Intercept	9.34	15.99	0.58	0.560	115	0.05	16.60	20.75	0.80	0.426	74	0.09
Cortisol (C)	-5.16	15.97	-0.32	0.747	115	0.03	-5.52	23.06	-0.24	0.812	74	0.03
Perceived Stress (PS)	0.12	0.77	0.16	0.874	115	0.01	1.03	1.02	1.00	0.320	74	0.12
Resiliency (R)	-0.50	3.65	-0.14	0.892	115	0.01	-1.39	4.73	-0.29	0.770	74	0.03
Baseline PTSD	0.69	0.11	6.32	<0.001***	115	0.51	0.42	0.16	2.55	0.013*	74	0.28
C * PS	0.69	0.79	0.87	0.387	115	0.08	0.59	1.04	0.57	0.572	74	0.07
C * R	1.34	3.55	0.38	0.706	115	0.04	1.57	5.00	0.31	0.754	74	0.04
PS * R	-0.02	0.17	-0.09	0.928	115	0.01	-0.17	0.23	-0.74	0.461	74	0.09
C * PS * R	-0.17	0.19	-0.88	0.379	115	0.08	-0.14	0.24	-0.60	0.553	74	0.07
	Depression Symptoms after 3 Months						Depression Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	-1.53	6.52	-0.23	0.816	105	0.02	2.26	8.49	0.27	0.791	72	0.03
Cortisol (C)	9.45	7.13	1.33	0.188	105	0.13	9.27	8.84	1.05	0.298	72	0.12
Perceived Stress (PS)	0.46	0.30	1.52	0.132	105	0.15	0.76	0.41	1.82	0.072	72	0.21
Resiliency (R)	1.01	1.47	0.69	0.490	105	0.07	-0.24	1.92	-0.12	0.902	72	0.01
Baseline Depression	0.51	0.11	4.52	<0.001***	105	0.40	0.15	0.18	0.83	0.407	72	0.10
C * PS	-0.30	0.34	-0.86	0.389	105	0.08	-0.30	0.44	-0.67	0.506	72	0.08
C * R	-2.24	1.57	-1.43	0.156	105	0.14	-1.75	1.95	-0.90	0.370	72	0.11
PS * R	-0.07	0.07	-1.05	0.295	105	0.10	-0.07	0.10	-0.78	0.441	72	0.09
C * PS * R	0.08	0.08	1.02	0.312	105	0.10	0.05	0.11	0.46	0.644	72	0.05
	Anxiety Symptoms after 3 Months						Anxiety Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	-8.91	8.94	-1.00	0.321	116	0.09	-9.78	11.72	-0.84	0.406	84	0.09
Cortisol (C)	-9.01	8.98	-1.00	0.318	116	0.09	-4.48	12.26	-0.37	0.716	84	0.04
Perceived Stress (PS)	0.58	0.41	1.42	0.158	116	0.13	1.25	0.56	2.22	0.029*	84	0.24
Resiliency (R)	2.25	2.01	1.12	0.264	116	0.10	1.93	2.62	0.74	0.463	84	0.08
Baseline Anxiety	0.63	0.10	6.08	<0.001***	116	0.49	0.52	0.15	3.53	<0.001***	84	0.36
C * PS	0.62	0.45	1.38	0.172	116	0.13	0.81	0.60	1.35	0.179	84	0.15
C * R	1.97	2.00	0.99	0.327	116	0.09	1.23	2.67	0.46	0.647	84	0.05
PS * R	-0.10	0.09	-1.09	0.278	116	0.10	-0.21	0.13	-1.63	0.107	84	0.18
C * PS * R	-0.12	0.11	-1.16	0.250	116	0.11	-0.21	0.14	-1.49	0.139	84	0.16
	Weekly Alcoholic Drinks after 3 Months						Weekly Alcoholic Drinks after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	28.30	35.38	0.80	0.426	104	0.08	17.47	19.27	0.91	0.367	75	0.10
Cortisol (C)	-14.29	36.65	-0.39	0.697	104	0.04	-5.34	19.74	-0.27	0.788	75	0.03

Perceived Stress (PS)	-0.77	1.55	-0.50	0.620	104	0.05	-0.22	0.85	-0.26	0.796	75	0.03
Resiliency (R)	-3.68	7.90	-0.47	0.642	104	0.05	-1.68	4.30	-0.39	0.697	75	0.05
Baseline Alcohol/Wk	0.60	0.18	3.34	0.001**	104	0.31	1.05	0.14	7.29	<0.001***	75	0.64
C * PS	0.29	1.80	0.16	0.872	104	0.02	-0.39	0.96	-0.41	0.683	75	0.05
C * R	2.14	8.11	0.26	0.792	104	0.03	1.00	4.29	0.23	0.816	75	0.03
PS * R	0.15	0.37	0.40	0.690	104	0.04	-0.01	0.21	-0.05	0.961	75	0.01
C * PS * R	0.02	0.43	0.04	0.970	104	<0.01	0.13	0.23	0.57	0.573	75	0.07
	Sleep Quality after 3 Months						Sleep Quality after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	1.99	5.76	0.35	0.731	84	0.04	11.35	6.94	1.64	0.107	58	0.21
Cortisol (C)	-3.17	6.13	-0.52	0.607	84	0.06	-7.20	7.52	-0.96	0.343	58	0.12
Perceived Stress (PS)	-0.15	0.27	-0.54	0.593	84	0.06	-0.35	0.34	-1.02	0.313	58	0.13
Resiliency (R)	-0.24	1.31	-0.18	0.856	84	0.02	-2.22	1.56	-1.43	0.159	58	0.18
Baseline Sleep Quality	0.79	0.10	8.10	<0.001***	84	0.66	0.72	0.13	5.64	<0.001***	58	0.59
C * PS	0.23	0.33	0.69	0.493	84	0.07	0.36	0.41	0.88	0.385	58	0.11
C * R	0.54	1.39	0.39	0.700	84	0.04	1.33	1.65	0.81	0.423	58	0.11
PS * R	0.05	0.07	0.69	0.493	84	0.07	0.09	0.08	1.09	0.280	58	0.14
C * PS * R	-0.03	0.08	-0.40	0.688	84	0.04	-0.06	0.10	-0.60	0.548	58	0.08

Note. Effect sizes (*r)* were calculated using the formula $\sqrt{(t^2 / (t^2 + df))}$; p * <.05; ** $p < .01$, *** $p < .001$

Testosterone and Resiliency Models

	PTSD Symptoms after 3 Months						PTSD Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	3.56	15.91	0.22	0.824	115	0.02	9.40	20.38	0.46	0.646	74	0.05
Testosterone (T)	-14.45	17.63	-0.82	0.414	115	0.08	3.65	23.07	0.16	0.875	74	0.02
Perceived Stress (PS)	0.49	0.72	0.68	0.498	115	0.06	1.42	0.97	1.47	0.145	74	0.17
Resiliency (R)	0.76	3.60	0.21	0.833	115	0.02	0.32	4.60	0.07	0.944	74	0.01
Baseline PTSD	0.69	0.12	5.87	<0.001***	115	0.48	0.44	0.17	2.58	0.012*	74	0.29
T * PS	0.51	0.62	0.82	0.414	115	0.08	-0.40	0.85	-0.47	0.639	74	0.05
T * R	3.27	3.90	0.84	0.403	115	0.08	-1.07	5.06	-0.21	0.833	74	0.02
PS * R	-0.10	0.16	-0.59	0.556	115	0.06	-0.27	0.22	-1.26	0.211	74	0.15
T * PS * R	-0.12	0.15	-0.80	0.425	115	0.07	0.11	0.20	0.55	0.586	74	0.06

	Depression Symptoms after 3 Months						Depression Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	-2.46	6.44	-0.38	0.703	105	0.04	0.13	8.42	0.02	0.987	72	<0.01
Testosterone (T)	12.36	6.93	1.78	0.078	105	0.17	4.68	9.41	0.50	0.621	72	0.06
Perceived Stress (PS)	0.50	0.28	1.78	0.079	105	0.17	0.89	0.38	2.33	0.023*	72	0.26
Resiliency (R)	1.19	1.44	0.83	0.409	105	0.08	0.32	1.88	0.17	0.864	72	0.02
Baseline Depression	0.58	0.11	5.20	<0.001***	105	0.45	0.14	0.16	0.92	0.363	72	0.11
T * PS	-0.58	0.24	-2.37	0.020*	105	0.23	-0.37	0.35	-1.06	0.292	72	0.12
T * R	-2.81	1.53	-1.84	0.069	105	0.18	-1.17	2.05	-0.57	0.569	72	0.07
PS * R	-0.08	0.06	-1.30	0.196	105	0.13	-0.11	0.09	-1.25	0.217	72	0.15
T * PS * R	0.14	0.06	2.43	0.017*	105	0.23	0.11	0.08	1.27	0.210	72	0.15
	Anxiety Symptoms after 3 Months						Anxiety Symptoms after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	-13.13	8.81	-1.49	0.139	117	0.14	-21.17	11.68	-1.81	0.073	84	0.19
Testosterone (T)	-17.68	9.84	-1.80	0.075	117	0.16	6.11	12.77	0.48	0.633	84	0.05
Perceived Stress (PS)	0.85	0.38	2.22	0.028*	117	0.20	1.94	0.53	3.66	<0.001***	84	0.37
Resiliency (R)	3.10	1.97	1.57	0.119	117	0.14	4.61	2.61	1.77	0.080	84	0.19
Baseline Anxiety	0.63	0.11	5.86	<0.001***	117	0.48	0.58	0.16	3.69	<0.001***	84	0.37
T * PS	0.45	0.35	1.30	0.195	117	0.12	-0.44	0.44	-1.00	0.320	84	0.11
T * R	3.99	2.17	1.84	0.069	117	0.17	-1.33	2.79	-0.48	0.635	84	0.05
PS * R	-0.16	0.09	-1.76	0.081	117	0.16	-0.39	0.12	-3.12	0.002**	84	0.32
T * PS * R	-0.08	0.08	-0.97	0.335	117	0.09	0.10	0.11	0.91	0.366	84	0.10
	Weekly Alcoholic Drinks after 3 Months						Weekly Alcoholic Drinks after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*
Intercept	31.63	35.48	0.89	0.375	104	0.09	28.35	19.00	1.49	0.140	75	0.17
Testosterone (T)	6.28	40.12	0.16	0.876	104	0.02	-2.69	21.96	-0.12	0.903	75	0.01
Perceived Stress (PS)	-0.89	1.48	-0.60	0.551	104	0.06	-0.85	0.81	-1.05	0.299	75	0.12
Resiliency (R)	-4.70	7.86	-0.60	0.551	104	0.06	-4.68	4.23	-1.11	0.272	75	0.13
Baseline Alcohol/Wk	0.61	0.18	3.46	<0.001***	104	0.32	1.06	0.14	7.56	<0.001***	75	0.66
T * PS	-0.24	1.39	-0.17	0.863	104	0.02	0.22	0.80	0.27	0.787	75	0.03
T * R	-0.19	8.91	-0.02	0.983	104	<0.01	2.52	4.84	0.52	0.605	75	0.06
PS * R	0.19	0.35	0.54	0.591	104	0.05	0.17	0.20	0.87	0.388	75	0.10
T * PS * R	-0.01	0.34	-0.03	0.977	104	<0.01	-0.16	0.19	-0.82	0.417	75	0.09
	Sleep Quality after 3 Months						Sleep Quality after 6 Months					
Predictor Variables	β	*SE*	*t*	*p*	*df*	*r*	β	*SE*	*t*	*p*	*df*	*r*

Intercept	1.28	5.80	0.22	0.826	84	0.02	10.54	6.89	1.53	0.131	58	0.20
Testosterone (T)	-2.32	6.90	-0.34	0.737	84	0.04	7.79	8.31	0.94	0.352	58	0.12
Perceived Stress (PS)	-0.05	0.24	-0.22	0.829	84	0.02	-0.22	0.30	-0.75	0.455	58	0.10
Resiliency (R)	-0.10	1.30	-0.08	0.939	84	0.01	-2.10	1.52	-1.39	0.171	58	0.18
Baseline Sleep Quality	0.79	0.10	8.30	<0.001***	84	0.67	0.66	0.12	5.50	<0.001***	58	0.59
T * PS	0.07	0.24	0.27	0.786	84	0.03	-0.26	0.31	-0.84	0.403	58	0.11
T * R	0.54	1.59	0.34	0.735	84	0.04	-1.40	1.89	-0.74	0.461	58	0.10
PS * R	0.03	0.06	0.44	0.661	84	0.05	0.07	0.07	1.02	0.311	58	0.13
T * PS * R	-0.01	0.06	-0.10	0.919	84	0.01	0.03	0.08	0.34	0.739	58	0.04

Note. Effect sizes (*r)* were calculated using the formula $\sqrt{(t^2 / (t^2 + df))}$; p * <.05; ** $p < .01$, *** $p < .001$

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